TM 8-285

WAR DEPARTMENT TECHNICAL MANUAL

TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

WAR DEPARTMENT • 15 APRIL 1944

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This manual supersedes TM 8-285, 27 November 1942; TC No. 86, and sec. II, TC No. 90, W.D. 1942; sec. IV, TC No. 27, W.D., 1943; and Circular Letter No. 9, Surgeon General's Office, 2 January 1943

TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

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For explanation of symbols see FM 21–6.

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SECTION I

GENERAL

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1. INTRODUCTION. a. Chemical warfare agents are used to produce casualties, to render food, water, and material unusable, to make terrain impassable or untenable, to provide concealment, and to start fires.

b. The scope of chemical warfare is broad. It aims at groups rather than individuals. Gas can penetrate dugouts, emplacements, and trenches. With the help of the airplane, chemical agents may produce casualties and destruction well back into the rear areas.

c. The purpose of this manual is to acquaint medical personnel with the treatment of casualties produced by chemical agents. Since medical units are themselves subject to chemical attacks, the medical officer must be familiar with the tactics of, and defense against, these attacks, as presented in FM 3–5 and FM 21–40.

2. CLASSIFICATION OF AGENTS. Chemical warfare agents are classified according to their physiological action, persistency, and tactical use.

a. Classification by physiological action. (1) Lung irritants (choking gases) primarily irritate and damage the respiratory tract. (Example: phosgene.)

(2) Vesicants (blister gases) injure the eyes, produce reddening and blistering of the skin, and when inhaled damage the respiratory tract. (Example: mustard.)

(3) Lacrimators (tear gases) act primarily on the eyes, causing tears and intense, though temporary, pain. (Example: chloracetophenone.)

(4) Irritant smokes or sternutators (vomiting gases) irritate the nose, throat, and eyes. They may produce temporary prostration. (Example: diphenylaminechlorarsine (adamsite).)

(5) Systemic poisons (blood and nerve poisons) stop essential physiological processes. (Example: hydrocyanic acid.)

b. Classification by persistency. The ability of an agent to maintain an effective concentration under field conditions is called its persistency.

(1) Persistent agents maintain effective concentrations longer than 10 minutes, and then may last for days or weeks. They are used to neutralize or force evacuation of certain areas. (Example: mustard.)
(2) Nonpersistent agents maintain effective concentrations less than 10 minutes. They do not render ground untenable after the cloud has passed. (Example: phosgene.)

c. Classification by tactical use. (1) Casualty agents injure personnel. (Example: mustard and phosgene.)
(2) Harassing agents force the wearing of masks and thus impede operations. (Example: irritant smokes.)
(3) Screening agents produce obscuring smoke to prevent observation. (Example: white phosphorus.)
(4) Incendiaries ignite matériel and produce burns on personnel. Example: thermite.)

3. FUNDAMENTALS OF FIRST AID AND TREATMENT. a. Prompt first aid is the most important factor in reducing the number of casualties. It is concerned with the removal and neutralization of the chemical agent before serious injury occurs. Unless incapacitated, each man will care for himself.

b. Treatment, designed to promote healing after injury has occurred, should be distinguished from first aid. Treatment is the function of the Medical Department.

SECTION II

LUNG IRRITANTS

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4. GENERAL. a. The primary lung irritants are phosgene, chlorpicrin, chlorine, and nitrous fumes. In general, the gases which are the most irritating, such as chlorine and chlorpicrin, are most likely to injure the trachea and bronchi. Those which are less irritating, such as phosgene and nitrous fumes, produce their major effect on the lungs and cause pulmonary edema. In addition to the above-designated lung irritants, vesicants and certain systemic poisons also damage the respiratory tract. (See secs. III and VIII.)

b. Troops exposed to a lung irritant gas need not be withdrawn during combat unless signs of pulmonary distress are apparent. The surgeon should so advise the responsible field commander.

c. The service mask and collective protector offer adequate protection.

5. PHOSGENE (CG). a. Pathology. Phosgene has no effect on the skin. In the upper respiratory tract and the eyes it may produce a mild inflammatory reaction. Its main sites of action are the bronchioles and alveoli. Congestion, edema, and mild cellular infiltration of the bronchiolar or alveolar wall, with alternating patches of emphysema, are present soon after exposure. The permeability of the capillaries is increased, and plasma-like fluid escapes into the alveoli. This accumulation of fluid reduces the space vital for respiratory exchange, fills many of the bronchioles, and produces pulmonary consolidation. Areas of lung not so consolidated become emphysematous. As plasma is lost into the lungs, hemoconcentration and anoxemia result. Small hemorrhages, due to the anoxia, are frequent in the lungs and elsewhere, especially in the central nervous system. Death may result finally from oxygen want. If recovery takes place, the fluid in the lungs is absorbed or expectorated. Patchy areas of fibrosis represent the healed lung lesion. This focal scarring follows the injury

by the gas and usually is not a result of a bacterial bronchopneumonia. Occasionally, however, a bacterial pneumonia may be superimposed.

b. Symptoms. Immediately after exposure there is likely to be coughing, choking, a feeling of tightness in the chest, and lacrimation. The presence or absence of these symptoms is of little value in immediate prognosis, as some patients with severe cough fail to develop serious lung injury, while others with no signs of respiratory tract irritation go on to fatal pulmonary edema. There may be slowing of the pulse initially, followed usually by an increase in rate. This transient bradycardia is presumptive evidence of phosgene poisoning. A period follows during which abnormal chest signs are absent and the patient may be symptom-free. This interval commonly lasts 2 to 24 hours, but occasionally is shorter. It is terminated by the signs and symptoms of pulmonary edema. These begin with rapid, shallow breathing, painful cough, and *cyanosis* (blue stage). Nausea and vomiting may appear. As the edema progresses, discomfort, apprehension, and dyspnea increase and much frothy sputum is raised. Râles and rhonchi are audible over the chest. The patient may develop a shocklike state, with leaden, clammy skin, low blood pressure, and a feeble heart (gray stage).

c. Diagnosis. Irritation of the nose and throat by phosgene may be mistaken for an upper respiratory tract infection. Difficulty in breathing and complaint of tightness in the chest may suggest an acute asthmatic attack. The pulmonary edema is like that produced by many other war gases and may be confused with the edema associated with heart failure. Diagnosis can be established with certainty only from a definite history of exposure to phosgene.

d. Treatment. (1) REST. Pending the appearance of definite symptoms, men may continue their duties. When symptoms appear, if there is no respiratory distress with moderate exertion, casualties may be evacuated by walking. Essential equipment may be carried. Unnecessary exertion is to be avoided.

(2) WARMTH. Phosgene casualties should be kept only comfortably warm.

(3) OXYGEN THERAPY. Anoxia should be treated with oxygen. The need for oxygen is indicated by cough, dyspnea, cyanosis, and restlessness. Oxygen should be administered in as high a concentration as possible, in any case high enough to eliminate cyanosis. Oxygen decreases anoxia and quiets the patient. It is best administered by a mask which allows regulation of the proportions of oxygen and air. Lower and less well-controlled concentrations of oxygen are obtainable in tents and with nasal catheters. Carbon dioxide-oxygen mixtures are not indicated in phosgene poisoning.

(4) VENESECTION. There is at present no definite evidence that venesection is beneficial at any time; it is certainly harmful during the shock-like state.

(5) SEDATION. If oxygen fails to quiet the patient, morphine may be used subcutaneously in a dose of 0.01 to 0.015 gram (grains 1/6 to 1/4). The physician must weigh the value of its sedative effect against its depression of respiration. Codeine may be more useful than morphine, if cough is the prominent symptom. Sedative does of barbiturates are ineffective and larger doses may be harmful.

(6) CHEMOTHERAPY. Sulfonamides should not be given during the latent period. Thereafter, they should be administered for the prevention of pulmonary infection as soon as the edema begins to subside, as evidenced by an improvement in the patient's general condition. Two grams (grains 30) of sulfadiazine should be given initially, followed by 1 gram (grains 15) every 6 hours for 5 to 7 days. The urine should be kept alkaline during sulfonamide therapy by the administration of 2 grams (grains 30) of sodium bicarbonate every 4 hours. Sufficient fluids should be given to maintain a daily urinary output of at least 1 liter (about 1 quart).

(7) EXPECTORANTS. Expectorants should not be used in the treatment of pulmonary edema. They may be of value in relieving cough when irritation is limited to the upper respiratory tract.

(8) OTHER MEASURES. Atropine does not diminish edema or improve breathing; its acceleratory action on the heart is undesirable. Plasma is of no value in the treatment of phosgene poisoning. Infusions pass readily into the lungs and increase the edema. Concentrated plasma is even more harmful. Surgery, except emergency measures to save life, is contraindicated in the active stage of edema. If anesthesia is required, local infiltration or nerve block is the method of choice. Cardiac and respiratory stimulants, such as adrenalin, ephedrine, benzedrine, coramine, and metrazol, do more harm than good. Alcohol is contraindicated.

e. Convalescent care. Absolute rest must be continued until the acute symptoms have disappeared. As recovery progresses, exercise should be resumed gradually. Sitting in bed should be permitted first, then for brief intervals in a chair. Bathroom privileges should follow and then short periods of alternate walking and resting. Later the convalescent should walk increasing distances.

f. Prognosis. Prognosis should be guarded because of the insidious nature of the poisoning. Most deaths occur within the first 48 hours. The few which occur later are due largely to bronchopneumonia. Casualties from phosgene which survive more than 48 hours usually recover without sequelae. Rarely chronic bronchitis and bronchiectasis result. The incidence of tuberculosis is not greater in those poisoned by phosgene than in the general population.

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6. CHLORPICRIN (PS). a. Pathology. (1) Chlorpicrin vapor produces conjunctivitis and may, after severe exposure, cause corneal ulceration.
(2) The epithelium of the respiratory tract is injured by chlorpicrin. In the trachea and large bronchi the damage may be only slight and temporary, but in the small bronchi there is more necrosis of the epithelium than is produced by either phosgene or chlorine. In the alveoli, chlorpicrin produces less injury than phosgene but more than chlorine.
(3) The vapor of chlorpicrin irritates the skin and the liquid can produce deep burns.

b. Symptoms. Irritation of the eyes is the first symptom noted, as chlorpicrin is a strong lacrimator. This is frequently followed by pain in the chest, cough, nausea and vomiting. Severe exposure causes pulmonary edema, like that produced by phosgene, and generalized muscular weakness, with feeble heart action. Repeated small exposures increase susceptibility to asthmatic attacks from traces of this gas.

c. Diagnosis. Diagnosis can be established by a history of exposure, a characteristic flypaper odor on clothing, and the symptoms described in **b** above.

d. Treatment. Irritation of the eyes and nose can be relieved by irrigation with water, followed by the instillation of eye and nose drops (Medical Department item No. 91091). Inhalation of steam relieves the bronchial irritation. Codeine helps to allay the cough. If pulmonary edema develops, it is treated like that caused by phosgene.

e. Prognosis. Most deaths occur in the first 24 hours and if later are due usually to bronchopneumonia. Casualties surviving this period generally recover without sequelae.

7. CHLORINE (Cl). Chlorine is very irritating to the respiratory tract. It produces damage which may result in necrosis of the mucous membrane of the trachea, bronchi, and lungs. Pulmonary edema occurs, similar to that caused by phosgene. After exposure, the initial symptoms are: burning in the throat, violent coughing, and a feeling of suffocation. Pulmonary edema follows sometimes within 20 minutes. The history and the intense irritation of the nose and throat are aids to early diagosis. The treatment of chlorine poisoning is the same as that outlined for chlorpicrin. (See par. 6d.)

8. NITRIC (NITROUS) FUMES. a. Properties. Nitric fumes consist chiefly of mixtures of the oxides of nitrogen. They are orange-yellow to red-brown in color, soluble in water, and react with water and oxygen to form nitrous and nitric acids.

b. Occurrence of poisoning. The danger of nitrous fume poisoning is great if high explosives, such as smokeless powder or cordite, are burned

or detonated in the absence of sufficient ventilation. This may occur in gun pits, armored vehicles, ship magazines and turrets, as well as in mining and tunneling operations. Nitrogen dioxide may be used as a war gas.

c. Pathology. Nitrous fumes, like phosgene, so damage the bronchioles and alveoli of the lung that pulmonary edema with progressive anoxemia and hemoconcentration develops. Inflammatory changes of the upper respiratory tract also are produced but are moderate. Bacterial bronchopneumonia or peribronchial fibrosis may be sequelae. In the brain, hyperemia and multiple small hemorrhages are common in fatal cases. Continued exposure to nitrous fumes may lead to chronic inflammation of the mucous membranes of the eyes and upper respiratory tract.

d. Symptoms. When inhaled, nitrous fumes may cause little or no discomfort. There may be coughing or choking, possibly followed by headache, nausea, and vomiting. In some patients central-nervous-system symptoms predominate. After a latent period of variable length the signs and symptoms associated with increasing pulmonary edema and anoxemia may appear.

e. Diagnosis. The diagnosis is made from the history, the symptoms described, and sometimes from the pungent odor or the yellow discoloration of the exposed mucous membranes.

f. Treatment. Treatment of casualties with symptoms of pulmonary irritation is the same as that outlined for phosgene poisoning. (See par. 5d.) The few cases with symptoms referable to the central nervous system either die quickly or, on removal to fresh air, recover spontaneously.

g. Prognosis. Fatal cases usually die within 48 hours. Bronchopneumonia and varying degrees of pulmonary fibrosis and emphysema often follow recovery from the acute stage.

SECTION III
VESICANTS

9. GENERAL. a. The vesicants act primarily on the eyes and skin. In addition, they damage the respiratory tract when inhaled and, when absorbed, they cause systemic poisoning. The nitrogen mustards and the arsenical vesicants are the most dangerous in this last respect.

b. Vesicants poison food and water and render other supplies dangerous to handle.

c. Casualties contaminated with vesicants endanger unprotected attendants. Those in contact with such patients should wear, at least, protective masks, impermeable aprons and gloves, and other protective clothing if the area is contaminated.

d. Special precautions must be taken in receiving contaminated casualties to prevent injury of others. These casualties should be undressed in the open to prevent vapor accumulation indoors. They should be kept separate from uncontaminated patients until decontamination is complete. Contaminated litters, blankets, and equipment should be left outdoors. It is necessary to decontaminate equipment and ambulances after transporting such casualties. (See par. 146*d*, FM 21–40.)

e. Identification of the agent is important in order to apply specific treatment.

f. The service mask protects only the face, eyes, and respiratory tract. The eyeshield protects the eyes from contamination by liquid, but not from vapor. Permeable protective clothing and protective ointment prevent the vesicant from reaching the skin and will be issued in the theater of operations.

10. MUSTARD (H). a. Properties. Mustard is an oily liquid slightly soluble in water, more soluble in fats and oils, and freely soluble in gasoline, kerosene, acetone, carbon tetrachloride, and alcohol. These solvents do not

destroy mustard. It is slowly absorbed by rubber gloves and rubber clothing, and so may contaminate the inner surfaces of these articles. Its odor is like garlic or horseradish. Mustard disappears slowly through evaporation and hydrolysis in moist ground. It can be destroyed rapidly by decontaminating chemicals and by boiling. The persistence of hazard from the liquid agent or its vapor depends upon the degree of contamination, the type of terrain, and the weather conditions. Examples of persistence are given in the following table for average summer weather on open grassy ground.

Degree of contamination (pounds of H per square, 100 by 100 yards)	Can be traversed—	Can be occupied—
1,000	After 6 to 18 hours	After 4 to 5 days.
250	After 1 to 6 hours	After 1 to 2 days.
25	Immediately	After 1 day.

The table assumes services dress (not impregnated) and no antigas precautions. The above estimates are for average open ground and would be multiplied by factors of two or four for wooded areas. In winter the persistence is two to five times as long as in summer. All values of persistence times are estimates and should be checked by gas detectors. (See par. 127, FM 21–40.)

b. Eye. (1) PATHOLOGY, SYMPTOMS, AND PROGNOSIS. (*a*) The eye is more vulnerable to mustard than either the respiratory tract or the skin. Eye lesions follow an exposure of 2 hours to a concentration barely perceptible by odor (0.001 mg. per liter). This exposure does not affect the respiratory tract or skin.

(*b*) A latent period of 2 to 36 hours follows mild exposure, after which there is lacrimation and sensation of grit in the eyes. The conjunctivae and lids become red and edematous. Heavy exposure irritates the eye after a latent period of minutes to hours, and produces more severe lesions. Mustard burns of the eye may be divided into groups.

1. Mild conjunctivitis (75 percent of cases in World War I). Recovery, 1 to 2 weeks.

2. Severe conjunctivitis (15 percent of cases). Blepharospasm, and edema of lids and conjunctivae. Orange-peel roughening of the cornea. Recovery, 2 to 6 weeks.

3. Mild corneal involvement (10 percent of cases). Areas of corneal erosion staining green with fluorescein, 2 percent. Superficial corneal scarring and vascularization. Iritis. Temporary relapses. Convalescence, 2 to 3 months. This group requires base hospital care.

4. Severe corneal involvement (approximately 0.1 percent of cases). Ischemic necrosis of conjunctivae. Dense corneal opacification with deep ulceration and vascularization. Convalescence, several months. Predisposition to late relapses.

(2) DECONTAMINATION OF EYES. Liquid mustard in the eye demands *immediate* irrigation or at the *earliest possible moment,* since irrigation is markedly effective in the first few seconds and worthless after 2 minutes. The eye must be flushed with water from the canteen or other uncontaminated source. The head is thrown back, the injured eye held open with the fingers, and water poured slowly into it. The eye should be rolled about during the washing, which should be continued at least 30 seconds and not over 2 minutes. Irrigation must be completed before the gas mask is put on, in spite of field concentrations of vapor.

(3) DECONTAMINATION OF EYELASHES AND LIDS. The lids, lashes, and skin areas close to the eyes are best decontaminated by washing with soap and water. Protective ointment is irritating to the eyes. If water is not available, liquid vesicants may be removed from the lids by dabbing carefully with a cloth or other absorbent.

(4) TREATMENT OF MUSTARD CONJUNCTIVITIS. (*a*) Mild lesions require little treatment. Although they seldom become infected, 3 percent sodium sulamyd solution, two drops instilled every 4 to 8 hours, (Medical Department item No. 1K76525) has an analgesic action and aids in preventing sepsis. Two drops of eye and nose drops (Medical Department item No. 91091) may be instilled for added comfort. It the lids tend to stick together during sleep, sterile petrolatum (Medical Department item No. 91165) may be applied to the lid margins.

(*b*) Mustard conjunctivitis, with edema of the lids severe enough to obstruct vision, is alarming to the patient. The lids should be gently forced open to assure the victim that he is not blind. Pain may be allayed by eye and nose drops (Medical Department item No. 91091). To prevent infection, a few drops of 3 percent to 10 percent solution of sodium sulamyd may be instilled every 4 hours, after the first 24 hours. A sulfonamide ophthalmic ointment may be used instead. The eyes must not be bandaged nor the lids allowed to stick. The accumulation of secretions in the conjunctival sac, or any pressure on the eye at this stage, predisposes to corneal ulceration. Irrigations must be held to the minimum necessary to dislodge secretions, since excess fluid and trauma loosen the injured corneal epithelium. Isotonic or slightly hypertonic sterile solutions must be used, never hypotonic solutions; 1 percent saline is satisfactory. When the lids can be opened sufficiently for an ophthalmic examination, the cornea should be stained with fluorescein, one drop of 2-percent solution in saline, or by the insertion of a fluorescein ophthalmic disk (Medical Department item No. 91163) into the lower conjunctival sac. The eye then should

be rinsed with a few drops of sterile saline and examined for yellowish-green staining of the cornea. Staining indicates a loss of corneal epithelium. If the cornea stains, or if iritis or photophobia is present, atropine should be administered in 1-percent solution, or as an atropine ophthalmic disk (Medical Department item No. 91163). The patient is then transferred to the care of the ophthalmologist. Sealing of the lids may be prevented by the application of petrolatum to the lid margins. Dark glasses or an eyeshade may be worn for photophobia, but should be discarded as soon as possible to prevent neurasthenia.

(5) TREATMENT OF INFECTED MUSTARD BURNS OF EYE. Secondary infection is serious. Secretions must be removed gently by a minimum of irrigation with 1 percent saline. The eye should then be filled with a 10-percent solution of sodium sulamyd every 2 hours, or a sulfonamide ophthalmic ointment every 4 hours. Irrigation should be employed only to remove the accumulated exudate. Local anesthetics should not be used unless necessary, and then not oftener than every 6 hours. It is very important to prevent sticking of the lids and the sealing in of purulent secretions. Persons developing corneal ulcer or other complications should be referred to the ophthalmologist.

c. Skin. (1) PATHOLOGY. (*a*) The severity of the lesions and the rapidity with which they develop are greatly influenced by weather conditions as well as by the degree of the exposure. Hot, humid weather strikingly increases the action of mustard. Even under temperate conditions, the warm, moist skin of the perineum, external genitalia, axillae, antecubital fossae, and neck are particularly susceptible.

(*b*) After a latent period of variable length an erythema gradually appears, resembling sunburn. Vesication may follow. Usually multiple pin-point lesions form and coalesce into a single large vesicle. The typical blister is very superficial, translucent, and surrounded by erythema. The blister fluid is clear, straw-yellow, and does not coagulate early. It is nonvesicant.

(*c*) The amount of dermal edema is variable. In severe burns it may be considerable, and limit motion of a limb. Large drops of liquid may produce a ring of vesicles surrounding a gray-white area of skin which, though necrotic, does not vesicate. If a blister is not ruptured, it begins to be resorbed in about week. The roof forms a crust, beneath which re-opidernization takes place. The vesicles frequently break, however, and infection may occur.

(*d*) Mustard burns usually are followed by a persistent brown pigmentation, except at the site of actual vesication, where there may be a temporary depigmentation.

(*e*) Repeated burns may lead to hypersensitivity of the skin to mustard.

(2) SYMPTOMS AND PROGNOSIS. Exposure of the skin is followed

by a symptomless latent period of hours or days which varies in length with the degree of contamination. In temperate weather this period usually lasts 6 to 12 hours. As lesions appear, the skin develops generalized itching, which may last 4 to 8 days or longer. Blisters usually appear within 18 to 36 hours. The uncomplicated mustard lesion heals without scarring, usually in 3 to 6 weeks.

(3) DIAGNOSIS OF SKIN LESIONS DUE TO MUSTARD. Nearly identical skin lesions are produced by mustard and the nitrogen mustards. Mustard burns are also very like those due to lewisite and other arsenical vesicants. Differentiation of mustard lesions from those produced by lewisite is based upon—

(*a*) History of exposure to mustard.

(*b*) Absence of pain or discomfort at time of contamination. (Lewisite is painful immediately.)

(*c*) Latent period before the development of symptoms (lewisite, none).

(*d*) Wide zone of erythema surrounding blisters (lewisite, not prominent).

It should be remembered that vesicular lesions, much like mild mustard burns, may be produced in sensitive individuals by a variety of substances, notably plant poisons such as poison ivy or poison oak.

(4) DECONTAMINATION OF SKIN. (*a*) Personal decontamination is an individual responsibility of all ranks in all branches. Only casualties who are unable to decontaminate themselves are cared for by the Medical Service.

(*b*) Personal decontamination is the removal of *liquid* mustard at the earliest possible instant. The importance of prompt action cannot be overstressed. Proper skin decontamination during the first minute is always successful. After 3 minutes on the hot sweaty skin, or 5 minutes on the cool dry skin, no method of decontamination will prevent blistering. Decontamination should be performed, however, no matter how late, as long as liquid mustard is still present. Decontamination is of no value after vapor exposure.

(*c*) The contaminated skin should be blotted quickly with the absorbent paper furnished with the ointment, or with a dry cloth. (Later destroy the used absorbent.)

(*d*) Protective ointment should then be squeezed onto the spot, or the hands, and thoroughly rubbed into the affected areas with the fingers for about 15 seconds. Excess ointment should then be wiped off. On large splashes, the ointment should be applied and removed once more.

(*e*) If reddening of the skin has appeared, cleanse the area with soap and water. Protective ointment is irritating to the reddened skin and should be used only when liquid mustard is still present and soap and water are not available for thorough washing. Solvents should not be used if soap and water are available.

(*f*) Should the supply of protective ointment run short, the following alternatives may be employed:

1. *Bleach paste.* Prepared by mixing one part bleach and two parts of water. Because of its irritant properties, it must be washed off the skin within 3 minutes.
2. *Solvents.* Any nonirritant organic solvent may be used in an emergency to dissolve and dilute the liquid mustard. Since solvents do not neutralize the vesicant, the mustard solution formed must be completely and rapidly removed from the skin by flooding with a large excess of the solvent. Alternatively, the area may be sponged repeatedly with cotton or gauze dampened with solvent, with care to avoid the spread of mustard from contaminated skin. Gasoline, kerosene, alcohol, and carbon tetrachloride (from automobile fire extinguishers) are most commonly available. (***Caution:*** Precautions against fire and explosion must be observed when employing inflammable solvents.)

(*g*) The decontaminated skin areas should be thoroughly washed with soap and water as soon as practicable after decontamination.

(*h*) Wounded cases, with liquid mustard contamination of the skin, will seldom be received at field installations in time to prevent subsequent blistering. Nevertheless, if erythema has not appeared, known or likely areas of contamination should be decontaminated as outlined above.

(5) DECONTAMINATION OF HAIR. The contaminated hair may be clipped off, or decontaminated with bleach paste. The scalp should then be washed with soap and water. Protective ointment may be used, but is difficult to apply and to remove.

(6) TREATMENT OF MUSTARD ERYTHEMA. Mustard erythema in mild cases requires little or no treatment. If annoying itching is present, considerable relief can be obtained with calamine lotion (Medical Department item No. 91050) containing 1 percent each of phenol and menthol. Severe erythema is often accompanied by edema, stiffness, and pain. Effective subjective relief and subsidence of edema may be obtained in these cases by application of a light dressing moistened with amyl salicylate (Medical Department item No. 91027). ***Caution:*** The strong odor of amyl salicylate may put the soldier at a disadvantage in close jungle fighting; the odor may assist the enemy in stalking him, especially at night. The dressing is remoistened every 12 to 24 hours for about 4 days. Amylsalieylate must not be used on face or genitalia. Painful erythema of the genitalia may be treated with the above calamine lotion and a suspensory bandage. Alternatively, petrolatum, a light protective dressing, and suspensory may be used.

(7) TREATMENT OF MUSTARD BLISTER. (*a*) Large blisters, if tense or painful, should be drained by puncture at the lower margin, after sponging the skin with alcohol. It is not necessary to express clots; they will be absorbed and will not delay healing. Blisters are often partially refilled with fluid 24 hours after drainage, when a second puncture may be desirable to prevent traumatic rupture and loss of blister top. Usually the blister (except on face or genitalia) may be covered with a dressing which can be moistened once or twice daily with amyl salicylate (Medical Department item No. 91027) if there is pain or itching. **Caution:** The strong odor of amyl salicylate may put the soldier at a disadvantage in close jungle fighting; the odor may assist the enemy in stalking him, especially at night. Thereafter, a dry-dressing or sterile petrolatum (or boric acid ointment) dressing may be used. Frequent dressings are not desirable. In all cases with moderate to severe burns, prophylactic chemotherapy will be instituted. Sulfadiazine orally is the drug of choice (sulfanilamide may be substituted) with an initial dose of 4.0 grams (60 grains), and 1 gram (15 grains) every four hours thereafter. Sufficient fluids should be given to maintain the output of urine over 1500 cc. (1½ qts.) daily. 2 grams (30 grains) of sodium bicarbonate may be given every four hours to keep the urine alkaline.

(*b*) If the dressing sticks to the wound, care will be necessary to avoid pulling off the blister top. It is good practice to trim the edges of the adherent gauze, leave it in place, and put a fresh dressing over it. If necessary to examine the wound, the dressing can be soaked off.

(*c*) Small blisters may be covered simply with a light dry dressing, moistened once daily for 3 or 4 days with amyl salicylate if the burn is painful or itches. **Caution:** The strong odor of amyl salicylate may put the soldier at a disadvantage in close jungle fighting; the odor may assist the enemy in stalking him, especially at night. The dressing can be removed ordinarily in 2 or 3 weeks, when the area will be covered by a thin, pliable crust.

(8) TREATMENT OF DENUDED AREAS. (*a*) Blistered areas which have become denuded may be treated with sterile petrolatum or boric acid ointment as for thermal burns. Prophylactic chemotherapy will be instituted as outlined in paragraph (7) (a).

(*b*) Strong antiseptics and escharotics of all kinds are contraindicated.

(*c*) Sterile technique should be employed, including face masks, as for thermal burns. Frequent change of dressing is to be avoided.

(*d*) Occasional extensive granulating surfaces may require skin grafting. Multiple pinch grafts have proved successful.

(9) TREATMENT OF INFECTED MUSTARD BURNS. (*a*) Contamination of mustard burns with saprophytic bacteria is common, but

not serious. If there is no inflammatory reaction, the treatment is the same as for uncontaminated burns. (See (7) above.)

(*b*) Infected burns with an inflammatory reaction should be considered infected wounds. They may be treated locally by the use of sterile petrolatum or boric acid ointment with proper cleansing and drainage, but should not be treated with sulfathiazole ointment (Medical Department Item No. 91213, because large areas may be involved with danger of excessive absorption. Infection is best controlled by oral administration of a sulfonamide as outlined in paragraph (7) (a).

(*c*) Strong local antiseptics are contraindicated.

d. Respiratory tract. (1) PATHOLOGY. (*a*) Inhalation of mustard vapor causes irritation of the mucous membranes of the respiratory tract. Inflammatory changes may include necrosis with pseudomembrane formation and slough. A cast of the tracheo-bronchial tree may be formed.

(*b*) The pulmonary parenchyma, injured by mustard vapor, shows patchy emphyesma, congestion, and atelectasis. These changes are insufficient to cause anoxia, but they may be complicated by bronchopneumonia which is responsible for almost all the deaths following mustard. The mortality from mustard in the American Expeditionary Force, slightly more than 2 percent, was almost entirely from inhalation of vapor.

(2) SYMPTOMS AND PROGNOSIS. Respiratory tract lesions, like skin injuries, develop slowly and do not reach maximal severity for several days. Symptoms begin with hoarseness, which may progress to aphonia. A cough appears early and becomes productive. Fever, dyspnea, and moist rales may develop. The incidence of bronchopneumonia is high. Convalescence is slow, and cough may persist a month or longer. Milder symptoms, like hoarseness, last only a week or two.

(3) TREATMENT OF RESPIRATORY TRACT INJURY DUE TO MUSTARD. Mild respiratory tract injury, with hoarseness and sore throat only, usually requires no treatment. Cough may be relieved by codeine and pharyngitis with alkaline gargles. Relief from nasal inflammation may be obtained with eye and nose drops (Medical Department item No. 91091). Since severe respiratory tract injuries predispose to bronchopneumonia, the prophylactic oral administration of sulfadiazine or sulfanilamide (2 grams (grains 30) initially and 1 gram (grains 15) every 6 hours thereafter) is indicated. (See par. 5d (6).) Laryngitis and tracheitis should be treated by steam inhalations. Morphine or the barbiturates can be used to quiet the patient. Secondary bronchopneumonia should be treated like any other bronchopneumonia.

e. Systemic and gastro-intestinal. (1) SYMPTOMS. (*a*) Severe exposure of the skin to mustard may cause transient nausea and vomiting. The systemic reaction from large mustard burns is like that from thermal burns of comparable size.

(*b*) Ingestion of food or water contaminated by liquid mustard produces pain, diarrhea, and prostration. Mustard vapor does not significantly contaminate food or water.

(2) TREATMENT. Atropine may prove useful in reducing the gastrointestinal activity. Injury due to the ingestion of liquid mustard in food or water may require morphine and atropine for the relief of pain, and shock therapy for collapse. Bismuth subcarbonate (or subnitrate) can be used for diarrhea.

(3) PROGNOSIS. (*a*) A few deaths are recorded from the systemic effects of mustard absorbed through the skin in extensive burns. These casualties showed pronounced leukopenia, which may be regarded as a bad prognostic sign.

(*b*) Severe injury from ingestion of mustard is rare.

11. NITROGEN MUSTARDS (HN). a. General. The nitrogen mustards are oily, colorless, or pale yellow liquids, sparingly soluble in water but freely soluble in organic solvents. Some possess a faint fishy odor, while others are odorless. Their volatility varies with the particular compound. All are persistent, though not equally so. They are more readily hydrolyzed than mustard, but less so than lewisite. All their hydrolytic products, except the final ones, are toxic.

b. Decontamination. The contaminated areas should be washed with large quantities of soap and water or water alone. If soap and water are not available, decontamination procedures are the same as for mustard. (See par. 10b (2) and (3), c (4) and (5).) They must be instituted immediately to be effective. Since protective ointment merely dilutes the nitrogen mustards and does not destroy them, it is necessary to wash off the film of ointment with water or soap and water as soon as possible.

c. Eyes. (1) SYMPTOMS AND PATHOLOGY. Nitrogen mustards irritate the eye before the skin or respiratory tract. The irritation (caused by the nitrogen mustards) appears in a shorter time than that from mustard but not so early as that from lewisite. Mild or moderate exposure causes mild smarting and lacrimation within 20 minutes. Thereafter symptoms may wax and wane until they become persistent about 2½ hours later and reach their maximum in 8 to 10 hours. After more severe exposure, symptoms may begin immediately and progress for 24 hours or longer. Mild exposure produces erythema and edema of the palpebral and bulbar conjunctivae and superficial steamy haziness of the cornea. Irritation, lacrimation, deep eye pain, miosis, and photophobia are usually present. After more severe exposure the symptoms described above are followed by spotty hemorrhagic discolorations of the iris. The corneal epithelium begins to show a roughened, lusterless surface, with areas of punctate staining demonstrable by the instillation of fluorescein (Medical De-

partment item No. 91163). Severe exposure may cause the corneal epithelium to exfoliate. Slit lamp examination will reveal clouding and edema of the corneal substance extending deep below Bowman's membrane. Local necrosis of the cornea may rupture the globe.

(2) DECONTAMINATION AND TREATMENT. These are the same as for mustard. (See par. 10b.) In general, the symptoms and the lesions are more severe, requiring intensive and early treatment with atropine (Medical Department item No. 91163).

(3) PROGNOSIS. The prognosis in contamination with any liquid nitrogen mustard is serious, unless the agent is removed by irrigation within a minute or two. Mild injury progresses to complete recovery in about 2 weeks. Severe injury heals more slowly, requiring 9 to 12 weeks or longer. The cornea heals by vascularization, and the iris with discoloration and atrophy. Scarring may be expected. The degree of recovery is remarkable, although relapses may occur, as from mustard injuries.

d. Skin. (1) SYMPTOMS AND PATHOLOGY. The skin is more resistant than other tissues to the vapors of the nitrogen mustards and is less affected by them than by mustard. In mild exposures there may be no skin lesions. After severe exposure erythema appears earlier than is the case in mustard contamination. There may be irritation and itching as with mustard. Later, blisters may appear in the erythematous areas. Liquid nitrogen mustards are also vesicant. The blisters produced are more superficial and more rapidly formed, but are otherwise similar to those caused by mustard.

(2) DECONTAMINATION AND TREATMENT. For decontamination of the skin see b above. Since the absorption of the liquid nitrogen mustards through the skin is more complete than that of mustard, decontamination should be carried out as late as 2 to 3 hours after exposure, even at the expense of increasing somewhat the severity of the local reaction. Later treatment of skin lesions is like that for mustard burns. (See par. 10c (7), (8), and (9).)

(3) PROGNOSIS. Most blistered areas will heal in 2 to 4 weeks if infection is prevented. Occasionally, deeper burns require a longer time.

e. Respiratory Tract. (1) PATHOLOGY. The lesions caused by nitrogen mustards are similar to those caused by mustard. They decrease in severity down the respiratory tract from the point of entry. In the nose, larynx, and trachea, there may be marked swelling, erythema, and necrosis of the mucosa, followed by sloughing, hemorrhage, and fibrino-purulent exudation. The larynx is especially vulnerable. Edema and necrosis may lead to respiratory obstruction. In severe cases the damage may extend to the bronchioles and alveoli. Although pulmonary edema usually is not massive, secondary pulmonary infection is common.

(2) SYMPTOMS. The symptoms are much the same as those due to mustard, namely, delay in appearance, irritation of the nose and throat, hoarseness progressing to aphonia, a persistent cough, evidence of lung edema. Bronchopneumonia may appear after the first 24 hours.
(3) TREATMENT. The treatment of casualties with respiratory tract involvement is the same as for mustard. (See par. 10d(3).)
(4) PROGNOSIS. Mild tracheitis is likely to result in a cough which persists for several weeks. The prognosis is grave if there is severe respiratory tract involvement. Most late deaths are due to pneumonia.

f. Gastro-intestinal tract. Following oral administration or systemic absorption, the nitrogen mustards cause injury to the intestinal tract. Lesions are most marked in the small intestine and consist of inflammatory and degenerative changes in the mucosa. In animals severe diarrhea, which may be hemorrhagic, occurs. In man, the ingestion or parenteral administration of 2 to 6 milligrams causes nausea and vomiting.

g. Systemic effects. (1) PATHOLOGY. The most specific effects of the nitrogen mustards are on hematopoietic and lymphoid tissue. These follow absorption from intact skin, respiratory or gastrointestinal tract. In bone marrow the degenerative changes can be detected within 12 hours and may progress to severe aplasia. The thymus, spleen and lymph nodes involute rapidly with necrosis and phagocytosis of their lymphocytes. This injury is demonstrable in the blood through a transient leucocytosis of a few hours' duration, followed by severe lymphopenia, granulocytopenia, thrombocytopenia, and a moderate anemia. The blood picture may show little change for 5 to 10 days after exposure, at which time the white count may fall below 500 cells/ $mm.^3$ The various nitrogen mustards differ in their abilities to produce these changes.
(2) TREATMENT. The blood should be studied carefully and transfusions of whole blood given for thrombocytopenia or anemia. Vomiting or severe diarrhea may call for the replacement of fluid in addition to symptomatic treatment with sedatives, atrophine, and opiates. If these symptoms are prolonged, every attempt should be made to maintain an adequate nutritional status by intravenous infusion of glucose, amino acids, and plasma, and the parenteral administration of vitamins.
(3) PROGNOSIS. Leucocyte counts below 2,000 and great loss in weight probably point to a fatal outcome.
(4) DIAGNOSIS. Diagnosis is based upon a history of exposure, a faint fishy odor on the skin and clothing, and signs and symptoms characteristic of mustard exposure, which appear more rapidly.

12. LEWISITE (L). a. General. (1) Lewisite is an oily, colorless to light amber liquid, with a faint odor of geraniums. It is more volatile and less persistent than mustard, making it more effective in cold weather.

Lewisite is readily soluble in gasoline, kerosene, and alcohol. Although poorly soluble in water, it is rapidly hydrolyzed in contact with moisture. Lewisite oxide, one of the hydrolysis products, is vesicant and toxic and may contaminate ground for long periods. Lewisite, like mustard, penetrates fabrics and rubber, making it dangerous to wear clothing or rubber gloves previously contaminated.

(2) Lewisite, like mustard, injures the eyes, skin, and respiratory tract, and may produce systemic effects. In contrast to mustard liquid, lewisite causes stinging pain in 10 to 30 seconds, which increases in severity. The risk of burns from field concentrations of vapor is small. No decontamination or treatment is necessary following exposure to vapor unless pain is experienced. Then the procedures to be followed are those to be described under liquid lewisite. (See b(2), (3), and (4) below).

b. Eye. (1) SYMPTOMS, PATHOLOGY AND PROGNOSIS. Liquid lewisite causes severe damage to the eye. On contact, pain and blepharospasm appear instantly. Edema of the conjunctiva and lids follows rapidly and closes the eye in an hour. Inflammation of the iris usually is evident by this time. After a few hours the edema of the lids begins to subside, while haziness of the cornea develops and iritis increases. The corneal injury, which varies with the severity of the exposure, may heal without residua, may develop pannus formation, or progress to massive necrosis. The iritis may subside without permanent impairment of vision, if the exposure was mild, or after heavy exposure hypopyon may ensue, terminating in necrosis, depigmentation of the iris, and synechiae formation. Liquid lewisite instantaneously produces a gray searing of the cornea like an acid burn at the point of contact. Necrosis and sloughing of both bulbar and palpebral conjunctivae may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild lewisite conjunctivitis in man heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

(2) DECONTAMINATION OF EYES. Eyes contaminated with liquid lewisite require immediate treatment with BAL solution (eye solution M–1, Medical Department item No. 1K24810) or BAL ointment (Medical Department item No. 91028 or No. 1K24808). If BAL is used the first minute following contamination, the eye usually recovers in a few days. When used 10 minutes after contamination, the lesion requires several weeks to heal and ordinarily leaves permanent damage. BAL preparations exert little influence after 30 minutes. Eye ointment BAL (Medical Department item No. 1K24808) is available to all troops in the combat zones. It is issued in a 3-gram ophthalmic ointment tube with a break-off pin at the tip. It is to be carried in the same carton with the protective ointment, and is used as follows:

(*a*) Break the pin off the tip of the tube of BAL ointment.

(*b*) If the eye can be opened by traction on the lower lid, with the fingers squeeze the ointment directly into the injured eye and massage the lids gently.

(*c*) If the eye cannot be opened, apply the ointment to the lids and rub in as much as possible between them.

(*d*) Rub a small quantity on the lashes, lids, and skin around the eyes.

Caution: Neither BAL ointment nor solution should be put into the eye unless there is acute pain. BAL causes sharp stinging pain and blepharospasm in the normal eye. The discomfort lasts about 30 minutes, followed by irritation and redness for several hours. BAL has the opposite effect in eyes contaminated with lewisite, giving marked and rapid relief from pain. When the identity of the contaminating agent is in doubt, it is advisable to use BAL for any acutely painful war gas contamination of the eye. Hydrogen peroxide solutions *must not be used* in the eye; they are worthless to treat lewisite injury and are damaging to the cornea.

(3) TREATMENT OF LEWISITE CONJUNCTIVITIS. The treatment is like that for mustard. (See par. 10b(4) and (5).)

c. Skin. (1) SYMPTOMS. Stinging is felt in 10 to 30 seconds after contact with liquid lewisite. This increases in severity as the lewisite penetrates, and in a few minutes becomes a deep aching pain After five minutes contact, a gray area of burned epithelium is apparent much like an acid burn. Erythema and edema of the skin appear in about 30 minutes. The erythema is like that caused by mustard but is more painful. Itching and irritation persist only about 24 hours

(2) PATHOLOGY. Liquid lewisite acts more rapidly and produces more severe lesions of the skin than does mustard. Lewisite vapor, however, is distinctly less dangerous than mustard vapor. Contamination of the skin with liquid lewisite is followed in a short time by erythema. Vesication follows, and tends to cover the entire area of erythema, so that the red peripheral halo associated with the mustard lesion is seldom seen. The lewisite blister, often indistinguishable from the mustard blister, is steep-sided with a thick roof and contains slightly opaque yellow fluid. Microscopically, the roof exhibits more complete necrosis than does that of the mustard blister, a greater infiltration of cells within the vesicle, and an injury extending much deeper into the corium. The vesicle fluid contains a trace of arsenic, but is nontoxis and nonvesicant. Blisters are often well developed in 12 hours and are painful at first, in contrast to the relatively painless mustard blister. After 48 to 72 hours the pain lessens. Deep burns from lewisite rarely occur in man, because the pain on contact gives warning in time for decontamination. Such burns probably would occur only in the case of an unconscious victim. Lewisite can penetrate the skin, subcutaneous tissue, and muscle, causing enormous edema and gelat-

inous necrosis of the affected part. This is followed by failure of circulation, gangrene, and slough.

(3) PROGNOSIS. Lewisite erythema heals somewhat more rapidly than must erythema, and with less pigmentation. Small lewisite blisters heal in about the same time as those due to mustard. The larger lewisite lesions involve deep injuries which heal slowly and require skin grafts.

(4) DECONTAMINATION OF SKIN. (*a*) *Vapor.* The risk of skin burns from field concentrations of lewisite vapor is small and decontamination of the skin for such exposure should seldom be required. When drops of liquid lewisite contaminate the clothing, concentrated vapor from these drops penetrates the cloth and damages the underlying skin. Such clothing must be removed promptly. Decontamination of skin may be accomplished if specific measures are taken within a few minutes after contact. (See (*b*) below.)

(*b*) *Liquid.* The removal of liquid lewisite from the skin is the individual responsibility of all ranks in all branches. If the skin is wet with lewisite, the excess liquid is quickly removed by blotting with cotton or other absorbent. Protective ointment is then immediately applied as in contamination of the skin by mustard (see par. 10c (4) (*d*)).

(5) DECONTAMINANTS. BAL ointment or BAL solution (Medical Department item No. 91028 or 1K24810) are the best decontaminants for lewisite and other arsenicals. If available, they should be used.[1] The BAL is spread on the skin in a thin film, rubbed in with the fingers and allowed to remain at least 5 minutes. Thereafter the ointment or solution may be washed off when conditions permit. If protective ointment, or a BAL preparation, is not available, wash immediately with soap and water. Organic solvents are effective in preventing blisters only when used in the first few seconds. BAL preparations sometimes cause temporary stinging and itching urticarial wheals on the skin. The lesions usually last only an hour or so and should not cause alarm. Mild dermatitis, persisting a few days, may follow a single application. Dermatitis is fairly frequent if repeated applications are made to the same skin area. This prevents the regular use of BAL ointment as a protective film.

(6) TREATMENT FOR WOUNDED. Wounded men, contaminated with liquid lewisite, will seldom be received at field installations in time to prevent blistering. However, their burns may be lessened and significant systemic protection obtained if the decontamination procedures outlined in (4) and (5) above are carried out promptly.

(7) DECONTAMINATION OF HAIR. Contaminated hair may be clipped off or decontaminated with BAL solution or ointment and then washed with soap and water.

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[1] An individual issue of eye ointment BAL (Medical Department item No. 1K24808) is now being made to troops in combat zones.

(8) TREATMENT OF LEWISITE ERYTHEMA. The treatment of lewisite erythema is the same as that for mustard erythema, except that treatment seldom is required for longer than 24 hours (see par. 10c(6)). BAL ointment may be tried in the early stages.

(9) TREATMENT OF LEWISITE BLISTER. Lewisite and mustard blisters are treated alike. (See par. 10c(7).)

(10) TREATMENT OF DENUDED AREAS AND INFECTED LEWISITE BURNS. The treatment of these lesions is the same as that for similar lesions due to mustard. (See par. 10c(8) and (9).)

(11) TREATMENT OF DEEP LEWISITE BURNS. Large burns may be accompanied by serious systemic poisoning and shock demanding general measures as well as local treatment. Morphine and splinting of the affected parts may be necessary for the relief of pain. When the burned tissue becomes gangrenous, it may be allowed to slough, or it may be excised.

d. Respiratory Tract. (1) SYMPTOMS. Lewisite vapor is highly irritating to the respiratory tract and quickly induces sneezing and coughing. This property and the strong smell of geraniums have so effectively warned of its presence that no severe respiratory injuries have occurred. Inhaled lewisite vapor produces lesions of the respiratory mucosa essentially similar to those produced by mustard. Edema of the lung often is more marked, and is frequently accompanied by pleural fluid.

(2) TREATMENT OF RESPIRATORY TRACT INJURY DUE TO LEWISITE. Since there have been no human respiratory tract injuries from lewisite, treatment is recommended solely from the results of animal experimentation. In general, the treatment is a combination of that for the systematic effects of lewisite (see e (3) below) plus that for mustard respiratory tract injuries. (See par. 10d(3).)

(3) PROGNOSIS. The prognosis in respiratory tract injury from lewisite is unknown but probably is similar to that for an equivalent mustard injury, with the added danger of systemic arsenical poisoning.

e. Systemic. (1) PATHOLOGY AND SYMPTOMS. Liquid lewisite on the skin, as well as inhaled vapor, is absorbed and may cause systemic poisoning. A manifestation of this is a change in permeability which permits loss of sufficient fluid from the blood stream to cause hemoconcentration, shock, and death. In nonfatal cases hemolysis of erythrocytes has occurred with a resultant hemolytic anemia. Although lewisite is oxidized within the body, it may still be toxic. Its excretion into bile by the liver produces focal necrosis of that organ, necrosis of the mucosa of the biliary passages with peribiliary hemorrhages, and some injury to the intestinal mucosa. Acute systemic poisoning from large burns, in animals, causes

pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure.

(2) PROGNOSIS. Burns severe enough to cause shock and systemic poisoning are dangerous to life. Even though the patient survives the acute effects, the prognosis, must be guarded for several weeks.

(3) TREATMENT. There has been no experience in treating systemic lewisite poisoning in man, but the following measures may be of value:

(*a*) As soon as possible, apply the entire contents of one tube of BAL ointment (Medical Department item No. 91028) (22 grams) or one bottle of BAL solution (Medical Department item No. 1K24810) (eye solution M–1, 15 cubic centimeters) to the contaminated skin, spread widely, and rub in well to obtain the maximum absorption. Leave the preparation on the skin. Repeat at 12 hourly intervals for 48 hours and daily thereafter for 4 days.

(*b*) If signs of shock appear, administer the usual treatment, including plasma.

(*c*) Give fluids freely and intravenously if necessary.

(*d*) Give a high carbohydrate, high protein diet, employing intravenous glucose if the patient cannot retain food by mouth.

(*e*) Give supplementary vitamins in full dosage.

13. ETHYLDICHLORARSINE (ED). a. Properties. Ethyldichlorarsine is a colorless or brown liquid which is more volatile than lewisite and possesses a faint fruit odor.

b. Pathology. The lesions are the same as those caused by lewisite. (See par. 12a and b (1).)

c. Symptoms. Low concentrations of vapor produce no symptoms for the first minute. Stinging pain in the nose, a burning sensation in the throat, nausea, and vomiting then begin. Even though the gas mask is put on at once, symptoms may increase for several minutes. High concentrations are instantly so irritating to the eyes and respiratory tract that they compel wearing of the gas mask. A stinging and burning sensation is felt on the skin within 1 or 2 minutes. In very hot weather this may progress to redness in 10 minutes, and to shallow blistering in a few hours. Pain persists only about 24 hours, and the blisters crust over in a few days and heal rapidly. Liquid ethyldichlorarsine, like lewisite, is immediately painful on the skin and causes severe blistering. It produces eye injuries similar to but less severe than those due to lewisite.

d. Diagnosis. The following factors should be considered in making the diagnosis:

(1) History of exposure.

(2) Fruity odor of skin and clothing.
(3) Intense sternutatory irritant, and early vesicant effect.

e. Decontamination. Decontaminating procedures are identical with those for lewisite. (See par. 12b(2) and (3); c(4), (5), and (6).

f. Treatment. Treatment of mild respiratory tract irritation is the same as that for DM (see par. 18d). After decontamination, eye and skin lesions are treated as those due to mustard. (See par. 10b(4) and (5); 10c(6), (7), (8), and (9). BAL preparations (Medical Department item No. 91028 or1K24810) may be used in the early stages of skin erythema due to ethyldrichlorarsine or after severe exposure to prevent or treat systemic or respiratory tract injury.

g. Prognosis. Respiratory tract irritation from low vapor concentrations subsides within an hour. Skin burns in general, heal more rapidly than similar mustard burns. Liquid ethyldichlorarsine contamination in the eye causes serious injury, possibly blindness, unless a BAL preparation is promptly administered. (See par. 12b(2) and (3).)

14. PHENYLDICHLORARSINE (PD). a. Properties. This agent, a clear viscid liquid, is less volatile than lewisite or ethyldichlorasine. It is readily hydrolyzed in water.

b. Action. Phenyldichlorarsine, when inhaled, is a strong sternutator and lung irritant. Eye injury produced is similar to that caused by lewisite. (See par. 12b(1).) On the skin the vapor or liquid is only slightly less vesicant than mustard or lewsite. If absorbed, phenyldichlorarsine may produce systemic poisoning.

c. Pathology. The lesions and the systemic effects produced by phenyldichlorarsine are essentially those of lewisite.

d. Symptoms. Irritation of the eyes, nose, and thoat is prominent. Symptoms referable to the lungs and skin are like those produced by lewisite. (See par. 12c(1) and d(1).)

e. Treatment. Treatment in general is the same as that described for lewisite. (See par. 12c, d(2), and e(3).)

15. MIXED BLISTER GASES. a. General. Arsenical vesicants, such as lewisite (L) or phenyldichlorarsine (PD) are often mixed with mustard. Such mixtures do not produce more severe lesions than either agent alone, but they tend to confuse and make diagnosis difficult.

b. Decontamination. (1) EYES. If the exposure causes severe eye pain and blepharospasm, it should be assumed that an arsenical blister gas is present, and the first aid measures for lewisite should be applied at once (see par. 12b(2)). If the contamination is nearly painless, immediate

irrigation with water from the canteen or other uncontaminated source is employed. (See par. 10b(2).)

(2) SKIN. Any excess of the liquid mixture is blotted from the skin at once. Protective ointment is then applied as described under mustard (see par. 10c(4)). The ointment is thoroughly removed and a BAL preparation, if available, is rubbed on as for lewisite. (See par. 12c(4) and (5).) This should be removed immediately and applied again.

c. Treatment. (1) EYES. Definitive treatment of eye injuries due to mixtures is like that for mustard injury of the eye. (See par. 10b(4).)

(2) SKIN. Definitive treatment of skin lesions produced by mixtures is like that described for lewisite burns. (See par. 12c(8), (9), (10).)

SECTION IV
LACRIMATORS

16. SYMPTOMS AND TREATMENT. a. The more important lacrimators are chloracetophenone (CN), chloracetophenone solutions (CNS and CNB), and brombenzyl cyanide (BBC).

b. General symptoms produced by the lacrimators include lacrimation, photophobia, and blepharospasm, some irritation of the nose and of the freshly shaven face. In hot weather moist skin will be irritated. In addition, chloracetophenone solutions CNS and CNB may cause some mild papulovesicular dermatitis, especially in warm weather, and occasional vomiting. Lacrimator casualties ordinarily do not require medical attention.

c. First aid and treatment. (1) FIRST AID. The mask should be put on and rapid breathing maintained to aid circulation of air in the mask. The eyes are kept open as much as possible. They should not be rubbed. If a liquid or solid agent has entered the eye, it should be promptly washed out with water from the canteen.

(2) TREATMENT. (*a*) *Eyes.* Lacrimators produce a marked but self-limited irritation of the conjunctiva. When liquid lacrimators are splashed into the eye, the action is corrosive and resembles the burns of a strong acid. The instillation into the eyes of a solution of (¼ percent) sodium sulfite, if available, dissolves and neutralizes the irritating agent. Eye pain may be treated by instilling eye and nose drops (Medical Department item No. 91091). The further treatment is symptomatic as for any other burns of the eye.

(*b*) *Skin.* For skin burns caused by the lacrimators a 4-percent solution of sodium sulfite in 50-percent alcohol is advised. This must not be used in the eyes. The further treatment is the same as for other burns.

SECTION V

VOMITING GASES (NOSE GASES, IRRITANT SMOKES, STERNUTATORS)

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17. GENERAL. a. The vomiting gases, diphenylaminechlorarsine (DM, adamsite), diphenylchlorarsine (DA), and diphenylcyanarsine (DC) are crystalline solids which are dispersed by heat as fine particulate smokes. DM smoke is canary yellow near the point of emission, while those of DA and DC are white; all are colorless when diluted with air. Low concentrations are effective and smell like burning fireworks.

b. These agents produce strong pepperlike irritation in the respiratory tract, most pronounced in the trachea and large bronchi. The onset of symptoms may be delayed for several minutes, especially with DM, and effective exposure therefore may occur before the presence of the smoke is suspected. If the gas mask is then put on, symptoms will increase for several minutes, in spite of adequate protection. The soldier may believe his mask ineffective, remove it, and be further exposed. This is disastrous if the smoke is immediately followed by a lethal gas.

c. Both the service gas mask and the collective protector offer adequate protection against these agents.

18. DIPHENYLAMINECHLORARSINE (DM) (ADAMASITE). a. Pathology. DM produces local inflammation of the nose and nasal accessory sinuses, throat, and eyes.

b. Symptoms. These consist of pain and a sense of fullness in the nose and sinuses, accompanied by a severe headache, intense burning in the throat, and tightness and pain in the chest. Irritation of the eyes and lacrimation are produced. Sneezing is violent and persistent and coughing is uncontrollable. The nasal secretion is greatly increased and quantities of ropy saliva flow from the mouth. Nausea and vomiting are prominent. Mental depression may be so marked that the individual will need to be restrained to prevent self injury.

c. Diagnosis. This is made from the history of exposure and the relatively rapid spontaneous improvement which occurs despite the original miserable appearance and condition of the individual.

d. Treatment. The mask must be worn in spite of nausea and salivation, but it may be lifted from the face during actual vomiting. Frequent inhalations of chloroform (Medical Department item No. 91058), administered early, give relief. Aspirin may be given to relieve the headache and general discomfort. Few cases should reach the medical service for treatment, since recovery is prompt and the soldier can carry out his battle mission in spite of sternutators.

e. Prognosis. Ordinarily all symptoms disappear in about 1 or 2 hours. No permanent injury occurs even in severely affected individuals.

19. DIPHENYLCHLORARSINE (DA), DIPHENYLCYANARSINE (DC). The pathology, symptoms, diagnosis, treatment, and prognosis are similar to those of diphenylaminechlorarsine (DM). (See par. 18.)

SECTION VI

SCREENING SMOKES

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20. General. The most important of these agents are HC mixture (HC), sulfur trioxide-chlorsulfonic acid (FS), and titanium tetrachloride (FM). These smokes are not toxic in field concentrations, but may be dangerous in the heavy concentrations formed at the site of dispersion.

21. WHITE PHOSPHORUS (WP). See paragraph 28.

22. TITANIUM TETRACHLORIDE (FM). a. Pathology. The liquid produces acid burns.

b. Symptoms. Smoke generated by liquid FM is unpleasant to breathe as it irritates the nose and throat, but it is not dangerous in field concentrations. Exposure of the eyes to spray will cause conjunctivitis, with lacrimation and photophobia. Skin burns like those from acids are produced by contact with the liquid.

c. Treatment. The burned eyes or skin should be thoroughly washed with water and then treated like any other burn.

d. Prognosis. Good.

23. SULFUR TRIOXIDE-CHLORSULFONIC ACID SOLUTION (FS). a. Pathology. Acid burns are produced by contact with the liquid.

b. Symptoms. These are usually limited to a prickling sensation of the skin, but exposure to heavy concentrations may result in severe irritation of the eyes, skin, and respiratory tract.

c. Treatment. The eye is irrigated with water at once. Fluorescein (Medical Department item No. 91163) will reveal corneal ulceration. For pain, eye and nose drops (Medical Department item No. 91091) may be instilled. The eye is then covered with a light pad. Skin burns should be washed with water and then with sodium-bicarbonate solution. Later treatment should be that employed for other burns.

d. Prognosis. The prognosis depends on the degree of corneal ulceration.

24. HC MIXTURE (HC). a. Toxicity. Field concentrations of this smoke are harmless, but dangerous to fatal levels may be encountered in confined, poorly ventilated spaces near the point of smoke production.

b. Pathology. HC smoke, if inhaled, damages the respiratory tract by the action of the contained zinc chloride. Following severe exposure a chemical pneumonia with pulmonary edema may develop as in phosgene poisoning.

c. Symptoms. When HC is breathed in high concentrations there is a feeling of suffocation, and some irritation of the nose and throat, with coughing and choking. Later, signs and symptoms of pulmonary edema may appear.

d. Treatment. Treatment is like that for phosgene poisoning. (See par. 5d.)

e. Prognosis. The prognosis depends on the severity of exposure and the extent of pulmonary damage.

SECTION VII
INCENDIARY AGENTS

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25. GENERAL. The principal agents of this group are thermite (TH), magnesium and its alloys, white phosphorus (WP), and combustible oils. All generate tremendous heat and can inflict severe burns. Chemical fire extinguishers containing carbon tetrachloride (pyrene) or liberating carbon dioxide should not be used in confined spaces to extinguish thermite and magnesium incendiary bombs. When carbon tetrachloride comes in contact with flame or a highly heated metal, a mixture of phosgene, chlorine, carbon monoxide, and hydrochloric acid is liberated. The service mask does not offer protection against carbon monoxide.

26. THERMITE (TH). Thermite incendiaries burn at the temperature of about 4330° F. and scatter molten iron. Frequently explosive charges are added and make control hazardous. The particles or iron that lodge in the skin usually produce multiple small but deep burns. The particles should be cooled immediately with water and removed. Thereafter the treatment is that used for other thermal burns.

27. MAGNESIUM AND ITS ALLOYS. Magnesium burns at a temperature of about 3630° F. with a scattering effect similar to that of thermite. Deep burns are caused by its particles, which, unless removed promptly, result in slow healing. Removal is usually possible under local anesthesia. When explosive charges have been added to magnesium bomb, the fragments may be embedded deep in the tissues, causing localized gas formation and tissue necrosis.

28. WHITE PHOSPHORUS (WP). Extensive burns may be produced by incandescent particles of white phosphorus. The burns are usually multiple, deep, and variable in size. The smoke is nontoxic. White phosphorus continues to burn unless deprived of oxygen. The burned areas should be

immersed immediately in water or covered with dressings soaked with water, urine, or any nonirritant aqueous solution. Immersion should be continued until a 5-percent solution of copper sulphate (Medical Department item No. 91075) is applied as a wet dressing. Copper sulphate forms a noninflammable coating of copper phosphide on the phosphorus particles. All particles should be removed under water, unless the copper sulphate solution has been applied. They may be located by their phosphorescence in the dark. Burning particles are recognizable by the evolution of smoke. It is well to debride the burn promptly, if the patient's condition will permit, in order to remove unnoticed bits of phosphorus. Following the removal of the particles the lesions are treated as thermal burns. Salves with an oily base should not be used during the first few hours, since phosphorus is soluble in oil and might be absorbed with resultant systemic poisoning. Otherwise, the risk of systemic effects from embedded particles is small. Copper sulphate is innocuous even in large burns.

29. OIL INCENDIARIES. Burns may be produced by flame throwers and by oil incendiary bombs which may also contain phosphorus and sodium. Lung damage from heat and irritating gases may be a complication added to the injuries from incediaries, especially in confined spaces. Pulmonary embolism by particles of coagulated plasma, formed by the intense heat in peripheral veins, has been described. Morphine should be given guardedly to patients with pulmonary complications. The treatment of burns caused by oil incendiaries is like that for other heat burns.

SECTION VIII
SYSTEMIC POISONS

30. GENERAL. Systemic poisons produce their effects after absorption into the body and cause little or no local injury. Hydrocyanic acid, cyanogen chloride, arsine, and hydrogen sulfide are included in this group.

31. HYDROCYANIC ACID (AC). a. Physical properties. Hydrocyanic acid is a colorless, highly volatile liquid which boils at 26° C. Its vapor is extremely nonpersistent, and has the odor of bitter almond. Aqueous solutions are weakly acid.

b. Pathology. Hydrocyanic acid acts by combination with an enzyme essential for oxidative processes of the tissues. The central nervous system, particularly the respiratory center, is especially susceptible to this interference, and respiratory failure is the usual cause of death. In high concentrations of hydrocyanic acid (10 mg per liter or more) the amount inhaled in a few breaths may be sufficient to cause immediate death without anatomical changes. After exposure to lower concentrations, death may be delayed for hours to days. Small areas of hemorrhage and softening, the more pronounced the longer the course, may be found in the brain in fatal cases.

c. Symptoms. The symptoms depend upon the concentration of the the gas and the duration of the exposure. In high concentrations there is increased depth of respiration within a few seconds; violent convulsions after 20 to 30 seconds; cessation of regular respiration in 1 minute; occasional shallow gasps, and finally, cessation of heart action several minutes after initial exposure. Following moderate exposures, vertigo, nausea, and headache appear very early and are followed by coma and convulsions. These may persist for hours or days and be followed by death or recovery. If the patient recovers, after prolonged symptoms there may be evidence of damage to the central nervous system, such as irrationality, altered reflexes, and unsteady gait, which may last months or longer. Mild

exposure may produce headaches, vertigo, and nausea, but recovery is complete.

d. Diagnosis. The diagnosis may be made from the history, the odor, and the rapid onset of symptoms.

e. Treatment. Under combat conditions, treatment may be difficult. When hydrocyanic acid is detected, the soldier must adjust his gas mask instantly and hold his breath while doing so. If he is capable of doing this quickly, the hydrocyanic acid already absorbed usually will be detoxified. If he is incapacitated, emergency treatment must be given instantly by the nearest individual. The gas mask must be applied and an ampule of amyl nitrite (Medical Department item No. 10690) crushed and inserted quickly under the facepiece. The patient should inhale the amyl nitrite for 4 to 5 minutes, when another ampule may be given. If the soldier is conscious, he will be faint and dyspneic from this therapy, and may attempt to remove his mask. Artificial respiration is given if breathing has ceased. Where available, sodium nitrite and sodium thiosulfate should be administered intravenously. Sodium nitrite (10-cubic-centimeter doses of 1-percent solution) should be injected slowly to a total of 50 cubic centimeters. If necessary, epinephrine should be employed to counteract excessive fall of blood pressure. Between the nitrite injections 20 cubic centimeters of 5 percent sodium thiosulfate should be given intravenously and continued, if necessary, to a total of 500 cubic centimeters. Should the patient become greatly cyanosed as a result of methemoglobin formation, blood transfusions may be given. In general, treatment should be continued as long as there is the slightest sign of cardiac activity.

32. CYANOGEN CHLORIDE (CC). a. Properties. Cyanogen chloride is a colorless liquid which boils at 15° C. yielding a volatile irritant vapor. Although only slightly soluble in water, it dissolves readily in organic solvents. (Very low concentrations (0.0025 mg per liter) are sufficient to produce lacrimation.)

b. Pathology. The acute toxicity of cyanogen chloride is similar to that of hydrocyanic acid. The respiratory center is at first stimulated and then rapidly paralyzed. Cyanogen chloride, like chlorine, damages the respiratory tract resulting in mild inflammatory changes in the bronchioles, and congestion and edema of the lungs. The edema may form more rapidly than in phosgene poisoning. Rarely bacterial bronchopneumonia may complicate the original chemical injury.

c. Symptoms. The signs and symptoms combine those produced by chlorine and hydrocyanic acid. Following exposure there is immediately intense irritation of the nose, throat and eyes, with coughing, choking, tightness in the chest, and lacrimation. Thereafter the exposed per-

son may become dizzy and increasingly dyspneic. Respiration fails rapidly followed by unconsciousness and death within a few minutes. Convulsions, retching, and involuntary urination and defecation may occur. If these effects, due to the CN ion, are not fatal, the signs and symptoms of pulmonary edema may develop. There may be persistent cough with much frothy sputum, râles in the chest, severe dyspnea and marked cyanosis. As in phosgene or chlorine poisoning, a shocklike state may develop.

d. Prevention. The gas mask protects for a limited period. On detection, instantly hold breath, apply mask and breathe out.

e. Treatment. Treatment is that outlined for both hydrocyanic acid and for chlorine poisoning. (See pars. 6d and 31e.) The predominant signs and symptoms determine therapy. Artificial respiration must be given in cyanogen chloride poisoning if breathing has ceased.

f. Prognosis. If death does not follow promptly from cyanide, the effects of chlorine on the respiratory tract must be considered. (See par. 7.)

33. ARSINE (SA). a. Properties. Arsine is a colorless, odorless gas, but when impure it may have a garliclike odor in high concentrations.

b. Pathology. The gas is absorbed from the respiratory tract into the blood and gives rise to intravascular hemolysis. This results in anemia, hemoglobinemia, and hemoglobinuria. Through the action of circulating arsine and its oxidation products there is serious disturbance of the tissue metabolism of kidney and liver. The kidneys show marked tubular change and numerous hemoglobin-albumin casts. Anatomical changes in the liver are less constant, but hepatitis and focal necrosis may be present. Jaundice is due to hemolysis or to liver damage or both. Death results from renal or hepatic failure, anemia, or a combination of these.

c. Treatment. Therapy consists of bedrest, whole blood transfusions, oxygen, mild diuretics, and parenteral glucose. Specific therapy for arsine poisoning is still in an experimental stage.

34. HYDROGEN SULFIDE. a. This colorless gas in a low concentration has the odor of rotten eggs. In high concentrations it may dull the sense of smell and be difficult to recognize. Hydrogen sulfide is nearly as toxic as hydrocyanic acid.

b. Pathology. Hydrogen sulfide produces inflammation of the eyes, nose, and throat, and in high concentrations paralyzes the respiratory center or causes pulmonary edema.

c. Symptoms. At first there is irritation of the eyes, nose, and throat. Panting respiration and loss of consciousness follow quickly. Convulsions often occur as respiration ceases. If the exposure is not enough to be rapidly fatal, pulmonary edema may develop.

d. Treatment. As heart action continues after respiration has ceased, immediate first-aid treatment may be life saving. This consists of removal of the patient from the contaminated atmosphere at once or adjusting his mask immediately, artificial respiration, and inhalations, if possible, of oxygen-carbon dioxide mixtures or oxygen alone. Treatment of pulmonary edema developing later is the same as that from phosgene (See par. 5d.)

e. Prognosis. Mortality from severe exposure is high. When there are symptoms of lung damage the prognosis is like that in phosgene poisoning. (See par. 5f.)

SECTION IX
INCIDENTAL GASES

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35. GENERAL. a. This group includes carbon monoxide and ammonia. These may be encountered in dangerous concentrations in confined or poorly ventilated spaces.

b. Protection against incidental gases. The service mask and collective protectors are not efficient against carbon monoxide or ammonia. Special canisters are available.

36. CARBON MONOXIDE. a. Physical properties. Carbon monoxide is a colorless, odorless gas, which is lighter than air, into which it diffuses rapidly.

b. Occurrence in military operations. Carbon monoxide is formed by gun blasts, bursting shells, and internal combustion engine exhausts. Dangerous concentrations are apt to occur in confined spaces such as tank landing craft, garages, poorly ventilated gun turrets or emplacements, and in mining operations.

c. Pathology. Asphyxiation is produced by the inactivation of hemoglobin through combination with carbon monoxide. The resultant anoxia produces nervous system changes. Post mortem examinations reveal little beyond the characteristic cherry red color of the blood and hemmorhages in the brain. The dissociation of carbon monoxide from hemoglobin may be hastened by oxygen with or without added carbon dioxide.

d. Symptoms. The symptoms progress from throbbing headache, vertigo, yawning and poor visual acuity, to the development of cherry red membranes, weakness and coma, subnormal temperature, feeble pulse and perhaps death.

e. Diagnosis. The diagnosis is made from circumstances of exposure and the appearance of cherry red cyanosis.

f. Protection. Adequate ventilation should be provided for all inclosed spaces where carbon monoxide may be produced. The safety of the air in the space may be tested by introducing a cage containing a small animal or bird. The air will be safe for man to breath as long as these remain unaffected.

g. Treatment. Remove to pure air, give oxygen or oxygen-carbon dioxide mixture, and artificial respiration is necessary. Rest, blankets, and warm drinks are indicated also. Blood transfusions are valuable in desperate cases.

h. Prognosis. The longer the period of coma the less the chance for recovery. Most mildly exposed individuals recover with early treatment. Tachycardia and dyspnea may continue for months and there may be central nervous system disturbances ranging from simple neuritis to mental deterioration.

37. AMMONIA. a. Physical properties. Ammonia is a colorless gas which is soluble in water and has pungent, characteristic odor.

b. Occurrence in military operations. This gas has not been used in warfare but may be encountered in industrial accidents and bombings involving refrigeration plants.

c. Pathology. Exposure to high concentrations of ammonia produces prompt and violent irritation of the eyes and respiratory tract. There may be spasm and edema of the glottis of even necrosis of the laryngeal mucous membranes. Pulmonary edema may develop as in phosgene poisoning and may be complicated by bronchopneumonia.

d. Symptoms. Inhalation of high concentrations produces violent, burning pain in the eyes and nose, lacrimation and sneezing, pain in the chest, cough, spasm of the glottis, and pulmonary edema. Often there is temporary reflex cessation of respiration, which with the spasm and edema may cause asphyxia. Concentrations of 0.1 percent are intolerable to man. Liquid ammonia is vesicant.

e. Treatment. First-aid treament consists of prompt removal to pure air, and artificial respiration. Inhalation of the fumes of weak acetic acid or vinegar may be of some benefit. Later measures are directed toward the prevention of pulmonary edema and the treatment of bronchitis and pneumonia. (See par 5d.)

f. Prognosis. The mortality is high following severe exposure, wtih lower concentrations recovery is usually rapid, although bronchitis may persist.

SECTION X
OXYGEN DEFICIENCY

38. GENERAL. The proportion of oxygen in the atmosphere may be reduced to a dangerous degree in closed or poorly ventilated spaces such as shelters, compartments, or underground tunnels, by human consumption, combustion by fire, or dilution with other gases such as carbon dioxide or methane. The absolute reduction in oxygen at high altitudes, while mainly of concern to aviation, may also deserve consideration in land operations in mountains. It is obvious that the service gas mask is of no value in oxygen deficiency.

SECTION XI
ANIMAL CASUALTIES

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39. GENERAL. a. The material on human casualties from chemical warfare agents is generally applicable to animals. Hence only facts important in the handling of gassed horses, mules and dogs will be stressed.

b. The prevention of gas casualties in Army animals is important to eliminate lost working days. Animal gas masks and leg covers offer the best individual protection. Animals, especially horses, may, if masked, withstand exposures to vesicant vapors without developing incapacitating injuries. Protection by covers against airplane spray may be required. The dog gas mask is practicable for protection against toxic vapors if the animals are not in combat. The feet and legs of dogs are particularly vulnerable to liquid vesicants. Animals should be prevented from drinking from water holes, trenches, or shell craters and from pasturing in areas which have recently been contaminated, until the water and forage are known to be suitable for consumption. Water in deep wells or large streams and lakes is usually safe.

40. LUNG IRRITANTS. a. General. The effects of lung irritants in animals are like those in men.

b. Phosgene. (1) SYMPTOMS. Cyanosis, so prominent in humans, is masked in animals.

(2) TREATMENT. Heavy work is dangerous, especially after pulmonary edema develops. Animals in shock should be kept comfortably warm. Oxygen therapy for animals is not practicable under field conditions. Venesection has not proved beneficial and is certainly harmful during the shocklike stage. Contraindications in treatment are the same as those for man. If the animal survives 4 days, recovery may be expected, unless bronchopneumonia supervenes.

c. Chlorpicrin. Chlorpicrin irritates the upper bronchi and trachea as well as the alveoli. Pulmonary edema may appear. The clinical picture and treatment are as described for phosgene. The gas mask protects.

d. Chlorine (CL). Chlorine causes bronchospasm and a choking cough. If death is not immediate from intense bronchial spasm, the later symptoms and the treatment are those described for phosgene.

41. VESICANTS. a. General. (1) The terms "blister" and "vesicant" agents are misnomers for animals since vesication does not occur. The vesicants, especially the arsenicals, contaminate forage, water, grain, and other supplies.

(2) Before treating vesicant casualties, veterinary personnel must be protected.

b. Mustard. (1) INJURY CAUSED BY MUSTARD. (*a*) Incapacitating injury to animals may result from liquid mustard sprayed from aircraft or splashed from shell bursts, or from contact with recently contaminated ground. Mustard vapor produces less injury to animals than to man. A long coat of hair does not prevent injury but does impede penetration of liquid and cause lateral spreading. This results in a shallow lesion with a short healing time. Usually 10 to 20 minutes after the application of a liquid vesicant to the hair there is an *erection of the hair coat in the vicinity of the drop.* This phenomenon persists for 1 hour or longer and may be important in diagnosis. Two or more hours after contamination, edema of the underlying tissue appears and may increase for 24 hours. These signs appear more slowly in cold weather. As the edema subsides, particularly on the body skin, superficial layers may exfoliate. Where contamination has been heavy an eschar forms and later sloughs leaving an area of ulceration. The majority of such burns heal in 6 to 8 weeks, but some may require 14 weeks or longer. When horses and mules traverse ground where droplets of mustard remain, they frequently develop filling edema around their pasterns and fetlock joints. This is most pronounced in the fleshy parts of the legs. The animal becomes lame. Edema fluid oozes through the skin and accumulates in the hair coat. The skin becomes pulpy and soft. Fissuring and ulceration may occur and be aggravated by motion of the limb. The fine skin of the hollow of the heel is particularly susceptible and infection may be expected. The horny hoof including the frog is sufficiently resistant to mustard as to require no protection. If a horse must pass through a freshly contaminated area, its legs should be protected by impermeable leggings, or by a suitable protective ointment.[1] It should be borne in mind that shell holes may contain liquid vesicants for weeks after contamination.

[1] Protective ointment M–4 is irritant to the horse's skin.

(*b*) The eyes of animals, especially the horse, are more resistant than man's to injury from mustard vapor. Injury is not produced with short exposures to field concentrations. However, serious eye injury may follow severe exposure. Droplets in the eye cause conjunctivitis, keratitis, and temporary blindness or necrosis of the cornea with permanent opacity. Eye shields which need not be airtight give horses sufficient protection against liquid vesicants. The dog's eye is less resistant than the horse's to mustard vapor. The dog gas mask protects the eyes.

(*c*) Under ordinary field conditions severe exposure to mustard vapor is necessary to cause respiratory tract injury in horses. Here again the dog is more susceptible. Edema, necrosis, and ulceration of the mucous membranes of the respiratory tract follow inhalation of fine mists of mustard in the immediate vicinity of an H shell burst. Signs may appear as early as 10 minutes after exposure. The edema of the air passages often causes an obstructive dyspnea. A bacterial bronchopneumonia may follow.

(*d*) Contaminated fodder and water, or licking contaminated parts, may produce ulceration and edema of the buccal membranes. When the gastrointestinal tract is involved, abdominal pain and diarrhea may result.

(*e*) Vapor injury may be severe from the evaporation of liquid mustard on equipment close to the skin.

(2) DECONTAMINATION AND TREATMENT. Because the hair coat of animals impedes penetration, effective prophylactic measures may be instituted later in animals than in man. The healing time is shortened in proportion to the speed with which decontamination is employed and some mitigation of the injury may be expected even after 1 hour. Bleach paste (1 part chlorinated lime in 3 parts of cold water) or calcium hypochlorite (in 8 parts of cold water) should be used to neutralize mustard. This must be worked into the skin carefully avoiding the eyes. Decontaminants are irritant to the animal's skin and should be washed off within 2 to 5 minutes. Decontamination may be accomplished also by vigorous scrubbing of the contaminated area with a 5-percent solution of potassium permanganate. Swabbing with soap and water or with solvents like gasoline and kerosene usually spreads the agent, causing a shallow but larger lesion. Treatment of vesicant burns will depend upon the severity of the injury and the site of the lesion. Frequently, sprayed droplets of vesicants remain on the surface of the hair and cause slight superficial skin injury from vaporization. In such instances the droplets should be first wiped away with absorbents to prevent the treatment agents from carrying the liquid into the skin. Since liquid vesicants may remain in the hair for days, decontamination procedures should always be carried out before attempt-

ing to treat lesions that have already developed. In treating developed burns, the objectives are cleanliness of the wound and reduction of infection, thereby promoting healing. Cod liver oil ointment, tannic acid, gentian violet (Medical Department item No. 12135), potassium permanganate (Medical Department item No. 13722), or other preparations may be of value. Slight injury to skin not exposed to friction of harness and saddlery seldom needs treatment. Amyl salicylate must not be used on animals as it, *per se,* produces vesication. Eyes contaminated with liquid mustard should be irrigated immediately with water. If the conjunctivae and lids become edematous, bathing with boric acid solution may be of benefit. Pain may be allayed by local anesthetics (1 percent butyn, Medical Department item No. 11085). Treatment of injuries to the alimentary and respiratory tracts is symptomatic. Prevention of secondary pneumonia should be attempted with one of the sulfonamide drugs. Steam inhalations may provide respiratory relief. Fresh air, warmth, and nursing are essential. Food should be offered from the floor to promote drainage of nasal secretions. Appetizing foods should be given.

c. Lewisite. (1) INJURY CAUSED BY LEWISITE. Although the sensitivity of animals is nearly the same to lewisite as to mustard vapor, the lesion produced by liquid lewisite appears earlier and is more severe than that produced by liquid mustard. Lewisite burns resemble those caused by mustard, but are immediately painful and cause restlessness. There may be systemic effects.

(2) DECONTAMINATION AND TREATMENT. Decontamination for the destruction or removal of lewisite must be initiated at the first opportunity. Scrubbing the contaminated skin with a 5-percent solution of potassium permanganate or an 8-percent solution of hydrogen peroxide will reduce the injury and lessen the danger of arsenical poisoning. Bleach as indicated for mustard is also beneficial. Treatment of the eye, including the use of BAL, is similar to that for man. Late treatment is symptomatic.

d. Ethyldichlorarsine. Injury to the skin by ethyldichlorarsine is less than with lewisite or mustard. Treatment is similar to that for lewisite.

e. Nitrogen mustards. (1) These agents are less damaging to the skin of animals than equal concentrations of mustard or lewisite. On the respiratory tract their action produces injury of severity equal to that of mustard. The eyes of the dog and horse may be more susceptible to these vapors than to those of mustard. Injury to the eye from liquid nitrogen mustards is more severe than from mustard. The treatment of animal casualties should proceed along the general lines advised for mustard. In

cases of eye injury, where there is miosis, atropine sulfate (1 percent) should be instilled until mydriasis is induced. The prognosis in contaminations of the eye with these agents in the liquid form is serious unless the agent is removed by irrigation with water within 1 or 2 minutes.

(2) Severe exposures, in laboratory animals, result in lesions of the nervous and hemopoietic systems and in the gastrointestinal tract. (See par 11f and g.)

(3) Protective devices, especially to guard the eye against liquid spray, are important in prevention of animal casualties from nitrogen mustards. The horse and dog gas masks are effective.

42. LACRIMATORS AND IRRITANT SMOKES. While these agents are intolerable to man in even very low concentrations, under ordinary field conditions they have little effect upon animals, especially horses. Severe irritation may result when a liquid lacrimator gets in the eyes. The treatment is immediate irrigation with water or sodium bicarbonate solution.

43. SCREENING SMOKES. a. White phosphorus. (1) EFFECTS. Burning particles of white phosphorus produce deep burns on contact with the skin. The smoke is nontoxic.

(2) TREATMENT. Smother the burn immediately with water or mud. Keep the burn under water until a 2- to 10-percent copper sulfate solution can be applied. This excludes air by forming a protective metallic coating on the phosphorous particles. The particles can then be removed with forceps or a hemostat. Further treatment is that for ordinary burns.

b. Sulfur trioxide-chlorsulfonic acid solutions (FS). It is believed that this smoke in field concentrations will not affect an animal's skin. Eye burns may follow exposure to irritating concentrations. Water or sodium bicarbonate solution may be used for eye irrigations.

c. Titanium tetrachloride (FM). The liquid may produce burns on the skin and in the eyes, but it is not so irritant as FS. Treatment is the same as for burns caused by FS.

d. HC mixture. Usual field concentrations are not irritating to animals, but higher concentrations may produce respiratory tract damage like that caused by the lung-irritant gases.

44. INCENDIARY AGENTS. Treatment of burns from incendiary agents is that used for any heat burn.

45. SYSTEMIC POISONS. a. General. These are agents which produce their effects after absorption. The most important are hydrocyanic acid, cyanogen chloride, and arsine. Protection requires an efficient type of animal gas mask or collective protector.

b. Hydrocyanic acid and cyanogen chloride. Hydrocyanic acid produces asphyxia of the tissues, especially the central nervous system, and paralyzes the respiratory center. Cyanogen chloride, in addition to producing cyanide effects, irritates mucous membranes and may cause lung edema. If the animal exposed to HCN is not killed immediately, it probably will survive without treatment.

c. Arsine. For effects of arsine see paragraph 33. Treatment is symptomatic. The animal gas mask gives adequate protection.

46. INCIDENTAL GASES. Exposure to incidental gases such as carbon monoxide, nitrous fumes, and ammonia is not expected in the field.

APPENDIX I

TREATMENT OF BURNS

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1. GENERAL. Burns, as discussed in this appendix, include all cases with damage of the skin and underlying tissues due to heat, chemicals, or electricity.

2. FUNDAMENTALS OF TREATMENT. a. The prevention and control of shock.

b. The relief of pain.

c. The prevention and control of infection.

d. The prevention of contracture and excessive scarring by proper splinting and early skin grafting.

3. TREATMENT. a. General. (1) Proper steps for the prevention or treatment of shock should be instituted. In the presence of extensive burns, quantities of plasma up to 12 units may be required in the first 24 hours. If available, concentrated normal human serum albumin in appropriate amounts may likewise be used. Transfusion of fresh whole blood is often needed to combat the rapidly developing severe anemia which follows extensive burns; when anemia exists, whole blood transfusion is particularly indicated as a preliminary to skin grafting. Parenteral fluid replacement other than that attained by means of plasma or whole blood transfusion should be accomplished by means of 5 percent glucose in sterile distilled water. The intravenous administration of sodium chloride solution should be reserved for those burn cases in which mineral depletion occurs, such as that resulting from persistent vomiting.

(2) In all cases with moderate to severe burns, prophylactic chemotherapy will be instituted. Sulfadiazine, administered orally, is the drug of choice (sulfanilamide may be substituted) with an initial dose of 4.0 grams (60 grains). Subsequent doses of sulfadiazine should be given only under the direction of a medical officer. Although sulfonamide therapy may serve

to prevent infection, great care must be exercised in employing such therapy in burn cases. The extensive fluid loss and possible kidney damage so common in these cases increase the danger of renal complications from sulfonamide therapy. Maintenance doses of sulfadiazine should be given in 0.5-gram (7½ grains) doses every 4 hours until such time as an adequate kidney function, a daily output of at least 1,500 cubic centimeters, can be demonstrated, under which circumstance the dosage may be increased to 1 gram (15 grains) every 4 hours.

(3) Prophylaxis against tetanus is indicated in all patients with second or third degree burns.

(4) Pain should be relieved by adequate doses of morphine. Pain resulting from an extensive burn can ordinarily be relieved by a dose of ½ grain (0.030 gram) of morphine. In the presence of pronounced anoxia, large doses of morphine are dangerous, and under such circumstances the dose should not exceed ¼ grain (0.015 gram).

b. First-aid or emergency treatment of burned area. (1) The burned surface will be covered with a liberal amount of sterile petrolatum, or, if this is not available, boric acid ointment. The burn should then be covered with strips of a sterile fine-mesh gauze (44-mesh gauze bandage is satisfactory). Over this should be added a smooth thick layer of sterile gauze dressing (large or small first-aid dressings are especially suitable for this purpose). Finally, a gauze of muslin bandage should be firmly applied over the dressings.

(2) Contamination of burned surfaces by organisms from the nose and throat is responsible for most of the more serious infections which subsequently develop. Therefore, to minimize contamination from this source, masking should be practiced by the surgeon and assistants. If masks are not available, mouths should be kept closed.

(3) The prompt administration of plasma, when feasible, constitutes an important element in the emergency treatment of burns.

c. Treatment of burned area when patient arrives where hospital facilities exist. The burned area will be treated as follows, using standard operating room technique with patient and attendants fully masked:

(1) In cases in which the burned surface appears clean, no further preparation should be done. The use of detergents, such as lard, washing, and debridement will be reserved for those cases in which the burned surface is grossly soiled. In these cases the burned area, and then separately the surrounding skin for a considerable distance, are to be carefully and gently cleansed using cotton or gauze, neutral soap, and water. Green soap and brushes will not be employed. In the debridement, loose shreds of epidermis will be removed and this material saved for bacteriologic study if feasible.

Small blisters should not be disturbed, but larger ones may be punctured without removal of the epidermis. Skin that gives evidence of irreparable damage through its full thickness should be excised. Evidence of irreparable damage to deeper layers of skin may not be apparent for several days, and excision in such cases should be done as a secondary procedure. The resulting wound should be handled like any other open surgical wound, primary grafting of skin being carried out, if conditions permit. General anesthesia should be avoided if possible and pain during the preparation and application of dressings should be controlled by morphine.

(2) Tannic acid and all other escharotics will not be employed in the treatment of burns. The burned area will be covered with sterile petrolatum or, if this is not available, with boric acid ointment. Strips of a fine-mesh sterile gauze (44-mesh gauze bandage is satisfactory) should be applied. Over this should be added a smooth, thick layer of sterile dressing; this may consist of gauze, absorbent cotton, cotton waste, or cellulose. The dressings should be held in place by an evenly and firmly applied bandage; stockinette or some form of elastic bandage is more effective than the ordinary roller bandage. The dressing should extend well beyond the burned area, and in cases involving the extremities, should include all of the extremity distal to the burn. The principle of infrequent dressings in the treatment of burns is especially desirable. For this reason, unless complications develop, the dressing should not be disturbed for from 10 days to 2 weeks. Immobilization of the part by splinting should be effected when feasible.

(3) One of the most important factors in preventing contractures and in obtaining an optimum functional and cosmetic result in burns is early epithelialization. For this reason, skin grafting of granulating surfaces should always be done as soon as practicable.

APPENDIX II

CARE OF CONTAMINATED CLOTHING AND EQUIPMENT AT MEDICAL INSTALLATIONS

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1. INTRODUCTION. In the event of gas warfare, due care must be exercised at Medical Department installations to prevent injury to patients and medical attendants from clothing, blankets, or other equipment which has become contaminated with blister gases. Proper steps must also be taken to obtain timely replacement of items made unusable by contamination, and to insure the salvage and decontamination of such equipment.

2. REMOVAL OF CONTAMINATED CLOTHING AND EQUIPMENT. a. Clothing and equipment contaminated with a blister gas should be removed from the casualty at the earliest practicable moment, with due regard for the general condition of the patient.

b. Casualties should not be evacuated from Medical Department installations in clothing or blankets known to be contaminated with a blister gas; to do so may result in severe skin burns by contact with the blister gas and in burns of the eyes and respiratory tract from vapors which accumulate in confined spaces such as ambulances or small rooms.

3. DISPOSITION OF CONTAMINATED CLOTHING AND BLANKETS. An area out of doors at a safe distance from the medical installation (preferably at least 100 yards downwind) should be designated as a clothing dump, and contaminated blankets and clothing (except impermeable aprons and rubber gloves) should be transferred to this dump as conditions permit. The dump should be clearly marked "Danger, Gas."

4. NOTIFICATION OF SALVAGE OFFICER. The responsible Medical Department officer should notify (by field message or otherwise) the most available salvage officer in his unit area, advising him of the existence

of the dump of contaminated clothing and blankets, its exact location, and approximate size.

5. REPLACEMENT OF CONTAMINATED BLANKETS. a. To prevent the supply of blankets becoming exhausted, it will be necessary that those lost by contamination be replaced.

b. An informal check on the number of contaminated blankets sent to the clothing dump should be kept, in order that the approximate number of replacements required may be known.

c. If conditions permit, replacements may be obtained by requisition through the normal channels of medical supply (for example, regimental medical supply officer). If the time factor or tactical situation does not permit replacement through normal channels of supply, replacement may be requested from the nearest source of supply with which the unit has contact (for example, collecting company).

d. Succeeding echelons in the chain of combat medical supply should request replacement of blankets promptly, as their supplies are displaced forward. This should operate as far to the rear as the medical depot for the area.

e. Since salvaged contaminated blankets will normally be turned over to quartermaster depots following decontamination, the medical depot should requisition the necessary replacements from the quartermaster depot serving the area.

6. APRON, PROTECTIVE IMPERMEABLE (MEDICAL DEPARTMENT ITEM NO. 99030). a. The apron, protective, impermeable (Medical Department item No. 99030) is intended for use by personnel of Medical Department field installations while treating and handling blister gas contaminated casualties. The apron is always worn in conjunction with complete permeable protective clothing (see par. 114*b*, FM 21–40) and impermeable protective gloves (rubber) (Medical Department item No. 99263). The gas mask is also necessary as a part of the complete protective outfit.

b. Litter bearers moving into dangerously contaminated areas should don the complete outfit described above before entering such areas. Aid station attendants and others should don the complete outfit prior to handling or treating contaminated patients and the apron should not be removed until the danger of contamination has been removed. If treatment of patients is hampered by the use of the impermeable gloves, such gloves may be removed with comparative safety after removal of all of the patient's heavily contaminated clothing, and the treatment continued wearing protective gloves (cotton). Contaminated aprons may be worn

with safety for many hours in conjunction with permeable protective clothing. However, aprons should be decontaminated after each day of wear, as prolonged contact with the liquid blister gases may have a deleterious effect on the coated fabric. The complete outfit should also be worn while decontaminating litters, ambulances, and other equipment which may have been contaminated in transporting casualties.

c. Before donning the apron, the adjustment of leg, waist, neck, and sleeve closures of the permeable protective clothing are inspected to determine if a protective gas seal is secured and the protective gloves (cotton) are pulled on with gauntlet drawn well up over the sleeve of the shirt. With the neck strap of the apron buttoned, the head is thrust through the opening made by the neck strap and the apron; the left arm is inserted in the respective sleeve, and then the right arm and the tie straps and neck straps are adjusted to obtain a comfortable fit. The gas mask is worn over the apron. Immediately prior to moving into a contaminated area or handling contaminated patients, the gas mask is donned and adjusted. The collar of the shirt is turned up, and the hood, having been previously buttoned to the back of the shirt, is adjusted over the mask. Impermeable gloves are put on prior to handling contaminated patients or material.

d. In removing the apron, the procedure is as follows: After removal of the impermeable gloves, the neck strap is unbuttoned with the left hand and the two carrier straps released so that the canister and carrier hang suspended. The tie straps are released and the right arm removed from the sleeve by inserting the gloved forefinger of the left hand under the elastic cuff of the other sleeve and pulling. This procedure is repeated using the right hand in removing the left sleeve. The apron now falls freely from the body. Care should be exercised in the removal of the apron so that contaminated surfaces of the apron are not permitted to come into contact with the clothing of the wearer or other individuals. Decontamination procedure should be applied as soon as practicable to contaminated articles of clothing and equipment.

7. DISPOSITION OF CONTAMINATED GLOVES AND APRONS. a. It will not ordinarily be possible for aid stations to decontaminate aprons (Medical Department item No. 99030) and rubber gloves (Medical Department item No. 99263) during combat operations. Hence gloves and aprons of aid stations which become contaminated during combat should be wrapped in impermeable paper (litter cover paper), if available, and sent by the most available means to the collecting company with which they are in contact, to be exchanged for fresh aprons and gloves.

b. The collecting company when operating under combat conditions will send contaminated gloves and aprons which are received from aid sta-

tions, plus those which become contaminated at the collecting station, by the most available means to the supply officer for the medical battalion (or regiment, etc.) for decontamination and exchange for fresh supplies.

c. Decontamination of impermeable gloves and aprons for the attached medical troops of the division and for the medical battalion will ordinarily be accomplished by the headquarters section of the medical battalion.

d. Other medical units are responsible for decontaminating their own impermeable aprons and gloves.

8. REPLACEMENT OF CONTAMINATED GLOVES AND APRONS. a. Upon the receipt of contaminated aprons and gloves from aid stations, the collecting company will make automatic replacements in kind to the installation from which the items were received by the most expeditious means available.

b. Upon the receipt of contaminated aprons and gloves, the headquarters section of the medical battalion will make an automatic replacement in kind, to the installation from which the contaminated items were received, by the most expeditious means available.

9. DECONTAMINATION OF BLANKETS AND PERMEABLE PROTECTIVE CLOTHING. a. Contaminated blankets and permeable protective clothing removed from casualties are removed from the clothing dump by direction of the salvage officer. Decontamination of the blankets and protective clothing is then performed by Quartermaster Corps laundry units.

b. If the tactical situation permits, contaminated permeable protective clothing of aid-station personnel may be disposed of through a dump and decontaminated by the Quartermaster Corps laundry units, just as in the case of such clothing removed from casualties. (See a above.) However, if this procedure is not practicable, permeable protective clothing can be decontaminated by laundering with GI or laundry soap in lukewarm water (not over 100° F.). The contaminated garments should be stirred (except woolens to avoid excess shrinkage) in the heated soapy water for at least 5 minutes. This should be followed by two additional washes and three rinses of 5 minutes each. If an odor of blister gas remains, the process should be repeated.

10. DECONTAMINATION OF APRONS. Impermeable aprons (Medical Department item No. 99030) may be decontaminated by *one* of the following methods:

a. Method I. The apron is immersed in plan water at a temperature just below boiling for a period of 1 hour. It is then dried in air and returned to service.

b. Method II (For limited areas only). All visible liquid contamination is wiped off the apron. The contaminated areas are then sponged with an approximately 30-percent aqueous slurry of bleaching powder (Chemical Warfare Service Issue) [1] after which the apron is hung up and the adherent solution allowed to dry on it. ***Caution:*** The apron must not be soaked in the bleach slurry, or the slurry, in sponging, allowed to come in contact with the stitched seams as this will weaken the cotton threads. Aeration of the apron must be continued at least 24 hours in warm weather, or from 2 to 3 days in cold weather. The apron is then rinsed, dried, and returned to service.

11. DECONTAMINATION OF IMPERMEABLE RUBBER GLOVES. The decontamination of impermeable rubber gloves (Medical Department item No. 99263) must be done carefully to prevent severe chemical burns when subsequently using them. Either of the following methods may be used:

a. Method I. The gloves are immersed [2] for 24 hours in a 30-percent aqueous slurry of bleach powder (Chemical Warfare Service issue),[1] which is kept warmed to a temperature of 85° to 90° F. (barely warm). The gloves are aerated for 3 days, allowing the adherent solution to dry on them. The gloves are then rinsed, dried, and returned to service.

b. Method II. The gloves are kept immersed [2] in boiling water for 2 hours, then dried and returned to service. This method causes some deterioration of rubber, and should be avoided in favor of Method I, if practicable.

12. DECONTAMINATION OF GAS MASKS, WEB, CANVAS, AND LEATHER EQUIPMENT. a. Emergency decontamination of gas masks, web, canvas, and leather equipment is accomplished by the use of protective ointment. All visible liquid contamination is first wiped off with rags, after which ointment is applied to the contaminated areas and allowed to remain for 15 minutes. The surfaces are then wiped *clean* with rags. To be most effective, the ointment should be applied within 3 minutes after contamination. Generally, it is not practicable to decontaminate more than 2 square feet of surface by this method. ***Caution:*** Do not use protective ointment on the lenses of gas mask because it etches them severely. Eyepieces will be decontaminated so far as possible by rubbing them with a cloth or absorbent paper.

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[1] Bleach powder (Chemical Warfare Service issue) is available at medical battalions and higher medical echelons. In combat, battalion medical sections may obtain bleach powder from the supporting collecting company.

[2] In immersing the gloves, care must be taken that the gloves are filled with the solution, and that they are kept below the surface of the slurry.

b. Gas masks, if lightly contaminated by vapor or droplets, should be removed from their carriers and aired in the sun and wind at every opportunity. Long exposure to heavy vapor concentrations and heavy liquid contamination will require scrubbing with soap and warm water (85° to 90° F.), to remove surface contamination, followed by aeration for a minimum of 5 days in warm weather to a period of 2 or 3 weeks in cold weather depending upon the temperature and degree of contamination.

c. Gas mask carriers, first-aid pouches, and other web and canvas equipment may be decontaminated by soaking in water, to which 2 ounces of sodium carbonate (washing soda) per 10 gallons of water have been added, for 1 hour at a temperature just below the boiling point. The items are then hung up to dry and returned to service.

d. Shoes, straps, and other leather equipment may be decontaminated by soaking in water heated to a temperature of about 122° to 131° F. (about as hot as the hand can stand), for 4 hours, then dried and returned to service.

APPENDIX III

CHEMICAL AGENT CONTAMINATION OF FOOD, FORAGE, AND GRAIN

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1. GENERAL. a. Contamination of foodstuffs by chemical warfare agents may occur from contact with vapor, sprays or splashes of liquid, or solid chemicals. Unprotected food, forage, and grain supplies may be so contaminated that their consumption produce gastrointestinal irritation or systemic poisoning. The vesicants and arsenicals are the most dangerous.

b. While field decontamination may be difficult, large stores of foods must not be hastily condemned until available means for decontamination have been considered. Scarcity of supplies may at times make reclamation necessary. Prompt segregation of the heavily contaminated portions may prevent or minimize contamination of the remainder. Generally, foods not especially packed in protective packages constitute the major difficulty. The present method of packing foods used by the Quartermaster Department for overseas minimizes the dangers of contamination. With such packaging, in most cases only decontamination of the outer packing is required.

2. NATURE OF CHEMICAL CONTAMINATION. The vesicants and chlorpicrin are readily soluble in fats. They will be absorbed by foods of high fat content, and because of diffusion throughout the material, it may be impossible to remove them. Coagulation of protein by agents which are acidic or acid formers in high protein foods may limit diffusion of the agent. Hydrolysis of acid-forming gases in foods of high water content causes decomposition products which render the food unpalatable. Foods of low water and fat content will be relatively less easily contaminated by chemical agents and less difficult to decontaminate.

3. RECLAMATION OF CONTAMINATED SUPPLIES. a. General. The most effective and practical measures for purifying food, forage, and grain

when contaminated with chemical agents include washing with water or 2 to 5 percent sodium bicarbonate solution, trimming of exposed surfaces, serating adequately, and boiling in water. These measures may be ineffective if the decomposition products are toxic, as in the case of lewisite. In general, food, forage, or grain exposed to low vapor concentrations of chemical agents can be reclaimed by these procedures. It is impracticable to reclaim provisions that have been heavily contaminated by liquid droplets of vesicant agents. Unpackaged foods on which chemical agents can be seen with the unaided eye should be considered spoiled and their purification impracticable.

b. Lung irritants. This group of agents offers relatively little danger to food products. With the exception of chlorpicrin, these decompose rapidly upon contact with the water in foods, to form comparatively harmless compounds which may alter the flavor. Decontamination can be accomplished by washing, supplemented, where possible, by aeration. Chlorpicrin is slightly soluble in water, and is soluble in fat and most organic solvents. Its removal from foods of low water and fat content can be accomplished by aeration.

c. Lacrimators and irritant smokes. (1) Large stocks of supplies, when protected by covers or packages, probably cannot be contaminated with a sufficient quantity of the lacrimators or irritant smokes to warrant their destruction. These agents are not easily decomposed by hydrolysis and it would be difficult to reclaim foods *heavily* contaminated by them.

(2) Dry provisions contaminated by lacrimators can be decontaminated by aeration.

d. Vesicants. (1) When contaminated with *liquid* mustard or a liquid nitrogen mustard, foods of high water or fat content are unfit for consumption and reclamation is not practical. When foods have been exposed to vesicant vapor, they can be reclaimed by washing with soda solutions and rinsing with clear water, intensive cooking, or in the case of dry provisions, by 24 to 48 hours' aeration. Lean meat can be reclaimed by boiling in water for ½ hour or more, or in the case of the nitrogen mustards, with a 2-percent solution of baking soda. The water must be discarded after boiling.

(2) Lewisite, ethyldichlorarsine, and phenyldichlorarsine readily hydrolyze to poisonous arsenical oxides. Foods contaminated with these agents cannot be reclaimed.

e. Screening smokes. (1) HC, FM, FS, and WP smokes are nontoxic. They may alter the taste of foods by acids produced on contact with moisture, but do no damage otherwise.

(2) Liquid FM (titanium tetrachloride) can be washed from foods. Liquid FS (sulfur trioxide-chlorosulfonic acid solution) is highly corrosive and forms strong acids on contact with moisture. It may render unfit

for use foods which cannot be washed readily. After trimming, washing, or cooking, if the food does not taste too acid, it is safe to use.

(3) Unburned particles of white phosphorus are poisonous and must be removed from foods. Fats and oils may dissolve poisonous amounts of the agent and should be discarded.

f. Other agents. Carbon monoxide, arsine, and hydrocyanic acid will have little effect upon food supplies. Hydrocyanic acid is water soluble and foods with high water content may become unfit for consumption after exposure to high concentrations of that agent.

g. Meat from gassed animals. It may be necessary to use animals for food after they have been exposed to liquid splashes of chemical warfare agents. Economics may justify the early slaughter of exposed animals *before the effect of such exposure is shown.* If such animals are slaughtered in an approved manner in the preliminary stages of poisoning and all tissues exposed to the gas (lungs, local areas) are discarded, there is no objection to the consumption of the meat, provided the animal passes an otherwise satisfactory meat inspection. This is true even of animals poisoned by arsenical agents, since the edible tissue will contain amounts of arsenic too small to be toxic. Organs such as the liver, brain, heart, kidney, and lungs will contain relatively more arsenic than the musculature, and should be discarded. The meat should be well cooked.

h. Forage and grain exposed to vapor contamination by chemical agents can be decontaminated by aeration. Supplies so treated, especially if mixed with larger amounts of uncontaminated supplies, produce no ill effects when fed to animals. Forage, which is heavily contaminated by liquid vesicants, especially arsenicals, should not be used.

4. PACKAGED AND STORED PROVISION. In determining the disposition of packaged and stored supplies which have been contaminated, consideration must be given to the nature of the contaminant as well as to the type of foodstuff and the security afforded by the packaging material. Some of these factors are outlined as follows:

a. Airtight bottles and sealed tins give complete protection against vapor and liquid.

b. Wooden barrels, well sealed for the exclusion of air, give complete protection against vapor and moderate amounts of liquid.

c. Wooden boxes, not sealed for the exclusion of air, give little protection against vapor or liquid.

d. Waxed paper boxes, well sealed for the exclusion of air, give good protection against vapor and fair protection against liquid.

e. Paper wrapping give poor protection against vapor and very little against liquid.

f. Foil and cellophane wrappings, sealed for the exclusion of air, give good protection against vapor and liquid.

g. Ordinary textiles in a single layer packaging give practically no protection against vapor and liquid.

h. Coverings of sod and earth give good protection against vapor and liquid.

i. Open shelters give protection against liquid sprays and splashes. Closed buildings give protection against both vapors and liquids.

j. Generally, double layers greatly increase the protective efficiency of packaging materials.

5. When it is necessary to store bulk food supplies which are poorly protected by packaging, measures should be instituted to make the storage space as gasproof as possible. The most vulnerable food should be placed in the least exposed positions, keeping in mind the fact that the vapors of chemical warfare agents are heavier than air and tend to accumulate in low places. In the field, tarpaulins covering food supplies give fairly good protection against vapor and liquid agents. Food supplies which have become contaminated should be handled only by those trained in decontamination methods and equipped with protective clothing and gas masks.

APPENDIX IV

DETECTION OF CONTAMINATED WATER AND ITS PURIFICATION

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1. GENERAL. Contamination of water supplies with chemical agents has been encountered rarely, but in those instances the percentage of casualties was high.

a. Methods for detecting. Methods for detecting chemical agents make it possible to determine safe and unsafe water. The purification of contaminated water is difficult and requires chemicals and equipment not regularly issued to troops. Transportation of water may be required. Purification should be resorted to only in extreme emergency.

b. Important agents. The vesicants and the systemic poisons, cyanogen chloride and hydrogen cyanide, are the agents most likely to cause casualties when introduced into water. It is considered improbable that toxic concentrations of heavy metals and alkaloids will be encountered.

2. TOXIC LIMITS. The toxic limit for lewisite is 20 ppm (20 mg/1) (10 ppm (10 mg/1) as AS_2O_3), provided the water is chlorinated by the standard procedure for bacterial purification and is used for not more than 1 week. Nitrogen mustards in concentrations of 10 ppm (10 mg/1) have produced vomiting in man but have not caused actual casualties. In higher concentrations they are extremely toxic. Mustard dissolves slowly in water but may be found floating in tiny globules, as a film on the surface or collected in pools on the bottom. Small droplets when fed with water to rats have produced perforating ulcers in the intestinal tract. The limits for cyanogen chloride and cyanide are 10 ppm (10 mg/1).

3. REACTIONS WITH WATER. The three vesicants, lewisite, mustard, and nitrogen mustards, all react with water to form hydrochloric acid and the hydrolysis product corresponding to the agent. Lewisite reacts with water practically instantaneously, forming the hydrolysis product lewisite

oxide, which is toxic and somewhat vesicant. Mustard reacts with water to form the nontoxic thiodiglycol. A solution containing 100 ppm (100 mg/1) mustard becomes nontoxic at the end of 1 hour. Some types of mustard contain a highly odorous compound which renders the water nonpalatable even after hydrolysis. Nitrogen mustards hydrolyze slowly to a nontoxic product. A solution containing 100 ppm may remain toxic for 4 to 6 days. Cyanogen chloride, cyanide, and heavy metal salts dissolve in water but do not react extensively with it.

4. DETECTION. a. Kit, water testing, screening, for detection of chemical warfare agents. The Medical Department issues a kit (Medical Department item No. 99310) for simple, rapid field tests of water for dangerous chemical contaminations. These tests ordinarily are performed in the combat zone at the Engineer Corps water supply points. Further tests of water furnished by water supply troops of the Engineer Corps are not required, unless chemical contamination during its transportation from supply points is suspected. In combat it may be impracticable, or impossible, to obtain water from Engineer Corps water supply points. If it becomes necessary to use other water, the unit surgeon will be responsible for determining the potability of water procured for the troops of his unit. Under such circumstances, the *kit, water testing, screening* (Medical Department item No. 99310) should be employed by the unit surgeon. Tests should be made of the *raw water,* prior to chlorination. If this is found to be free of contamination, it may be used *after the usual purification by chlorination to render it safe from bacterial pollution* (ch. 3, FM 8–40).

b. Description of water testing kit. For the sake of simplicity, analytical procedures have been developed to employ dry reagents which are furnished as tablets or pellets of proper size. Except for warming with the hand in some of the tests, no heat is required. The kit contains equipment for testing 15 samples of water. The reagents and equipment are packed in a pocket-sized container, approximately 5½ by 3¾ by 1¾ inches, divided into 10 compartments. The container is constructed of transparent plastic. The kit contains 2 test tubes, a chlorine demand assembly (QM 11968), a bottle and tube for the detection of arsenicals by a modified Gutzeit method, and 7 vials containing reagents and test papers. The vials are identified by letters printed on the paper liners. Their caps are made of colored plastic matching the color of the paper liners. A test tube brush and pipe cleaner are provided for cleaning the apparatus.

c. Purpose. (1) The field kit for testing water is designed as for reconnaissance. It is employed to screen out sources of water so contaminated that they cannot be rendered potable by customary field methods, such as chlorination in the Lyster bag.

(2) Negative tests indicate that the water is suitable for chlorination and may be used by troops, within the limitations.
(3) If any of the tests are positive, the water should not be used until a more complete analysis can be made.
(4) The main purpose of the kit is to detect contamination of raw water. It is not designed for use on treated water as the chemical reactions of water treatment invalidate the interpretations.

d. Analytical procedures. A booklet issued with the kit gives specific directions for each test. Nontechnical language is used and the reagents are referred to by the letters on the vials. These directions must be followed exactly. Briefly, the tests involve the following chemical processes:
(1) Arsenicals are converted to arsines through the action of hydrogen, produced by the action of sodium acid sulfate on zinc. The arsine reacts with a sensitized paper to produce a stain. This is sensitive to 5 ppm.
(2) pH is determined by indicator paper.
(3) Mustard is detected by means of the DB3 reagent; 5 ppm (5 mg/1) of nitrogen mustard can be detected. Cyanogen chloride produces a yellow color with this reagent.
(4) The chlorine demand or chlorine uptake is determined by means of halazone tablets and an otolidine testing assembly (QM 11968). This test determines the presence of a number of less important agents not specifically tested for.
(5) If no evidence of contamination is found, odor and taste can be tried with safety.

e. Interpretations.

Test	Contaminated water [1]	Noncontaminated water [2]
Arsenic test	Positive	Negative.
pH test	pH below	pH above 6.
Mustard test	Positive	Negative.
Chlorine demand	Positive	Negative.
Taste and odor	Positive	Negative.

[1] Water will be considered contaminated if one or more of the tests give results indicated in this column.
[2] Water will be considered suitable for 1 week after bacterial disinfection by usual methods, if all tests give results indicated in this column.

f. Limitations. (1) If the tests are carefully performed, the threat of serious casualties from contamination of the water with known agents will be avoided.
(2) The tests provided by the kit will not screen out traces which are harmless when the water is used for short periods of time. When arsenic is detected, even though the water is passed as safe by the kit (that is,

an arsenic content which gives a stain shorter than ¼ inch on the test paper), the water should be used for drinking and cooking purposes not to exceed *1 week,* because of possible cumulative effects.

(3) Water may pass the test for nitrogen mustards and still give temporary symptoms if consumed in large quantities. Hence the water should not be used without special purification (by Engineer Corps water supply troops) if even the faintest blue color develops in the nitrogen mustard test. When the result of the test is questionable, the amount of water permitted per man, at the first drinking, should be limited to ½ pint; if no symptoms of nausea or vomiting develop during the succeeding 2 hours, the water may be used freely thereafter.

(4) The tests provided by the kit are not quantitative, and will therefore not serve as a guide for the purification of field water supplies. More elaborate methods are available to the division medical inspector and the laboratory, Army.

5. ACTION REQUIRED IF WATER IS FOUND TO BE CONTAMINATED BY CHEMICAL AGENTS. Whenever positive tests are obtained with the kit, water testing, screening, the water will be considered contaminated and the following actions taken:

a. The unit commander will be notified that the water source is contaminated and is unfit for drinking purposes.

b. The unit commander will establish the necessary safeguards to prevent troops from drinking the contaminated water.

c. An alternative source of uncontaminated water should be sought and, if found, should be employed.

d. If a source of uncontaminated water cannot be found, consideration should be given to moving to a different location, or to importing purified water to the area.

e. In any event, the contaminated water should not be used by troops until it is purified by the water supply troops of the Engineer Corps.

f. Contamination discovered in otherwise suitable water should be reported as promptly as possible to the headquarters of the division, or other comparable command, so that the matter can be brought to the attention of the division medical inspector and the commanders of Engineer Corps water supply unit for necessary action.

6. SCALE FOR ISSUE OF WATER TESTING KITS. The water testing kit has been added to the contents of the kit, treatment of gas casualty (Medical Department item No. 97769) and therefore is available to battalion

medical sections and to medical units so equipped. It is available also on separate issue to Engineer Corps water supply units and to the medical inspectors of divisions and higher echelons. As the kit is expendable, the individual items of its contents are not supplied for refilling. When the contents have become exhausted, the complete kit can be replaced through the usual channels of medical supply.

7. USE OF SUSPECTED WATER. a. Water which is only slightly contaminated can be used for periods not to exceed 1 week after chlorination for bacterial pollution.

b. When suspected water is used, great care should be taken not to stir up material from the bottom as it may contain chemical agents when the water above does not.

8. PROCEDURE IN CASE OF HEAVY CONTAMINATION. When water is too heavily contaminated to pass the screen kit test, every effort should be made to secure another source or to have pure water supplied from elsewhere. To meet emergencies, Sanitary Corps officers are provided with equipment for making more complete analyses. The methods for treatment are outlined briefly below. Only trained personnel should undertake such procedures.

9. PURIFICATION OF CONTAMINATED WATER. a. Water must be withdrawn from the intermediate levels with minimum disturbance of the surface and no disturbance of the bottom.

b. Treatment of large volumes. (1) The contaminated water is pumped into a canvas reservoir and a quantitative analysis made by a Sanitary Corps officer.

(2) It is then treated with activated carbon (200 mesh) in the following doses:

(*a*) For lewisite, 30 ppm (30 mg/1) carbon for each ppm (mg/1) lewisite.

(*b*) For mustard, 30 ppm (30 mg/1) carbon for each ppm (mg/1) mustard.

(*c*) For nitrogen mustard, 60 ppm (60 mg/1) carbon for each ppm (mg/1) nitrogen mustard.

(3) The carbon and water are mixed for 20 minutes to insure complete absorption of the agent by the carbon.

(4) 175 ppm (175 mg/1) of coagulant is added to the carbon-dosed water, together with sufficient alkali to give optimal coagulation.

(5) After thorough, gentle mixing, the water is allowed to coagulate and clarify by sedimentation for 30 minutes.

(6) The supernatant water is filtered through the portable water purification unit, at normal rate 0 + 10 gpm, or preferably more slowly.
(7) The filtered water must be tested quantitatively to see that it meets the following requirements:

(*a*) Mustards, not more than 2 ppm (2 mg/1).
(*b*) Lewisite (arsenicals), not more than 20 ppm (20 mg/1).
(*c*) PH above 5.
(*d*) Chlorine demand, less than 5.
(*e*) No chemical odor or taste.

c. Treatment in Lyster bags. (1) When the portable water purification unit is not available, small volumes can be purified by using two Lyster bags.
(2) If testing equipment is available to identify the contaminating agents and determine their concentrations, add activated carbon in the dosages given in 9b(2) above to the water in one Lyster bag. If the identities and concentrations of contaminants are unknown, add 2 pounds of activated carbon.
(3) Stir for 20 minutes.
(4) Add 1 ounce of alum and sufficient alkali to give optimal coagulation. These chemicals should be dissolved separately in small volumes of water prior to their addition to the Lyster bag.
(5) After thorough, gentle mixing, allow to coagulate and clarify by sedimentation for 30 minutes.
(6) Siphon the supernatant to another Lyster bag (preferably through a filter).
(7) After testing to insure that the requirements of b(7) above are met, the water in the second Lyster bag must be chlorinated.

10. CHLORINATION OF CONTAMINATED WATER. Chlorine reacts with some of the chemical agents making it difficult to remove them by the activated carbon and alum treatment. Therefore, chlorination should be carried out only *after* filtration through the portable purification unit, and the chlorine feed-line must be connected to the effluent pipe from the filter. In the case of treatment in the Lyster bag, chlorine is added in the *second* Lyster bag. When contamination is suspected, no chlorinating compounds should be added until the water has been clarified.

APPENDIX V

MEDICAL DEPARTMENT ITEMS FOR FIRST AID AND TREATMENT OF CHEMICAL WARFARE CASUALTIES

	Medical Department item No.	Item	Unit	Quantity
1.	97764	Kit, first aid, gas casualty:		
	97663	Container, for kit, first aid, gas casualty	ea	1
	10690	Amyl nitrite, USP, 5 minim, USP amp. 10.	pkg	1
	1K24810	Eye solution BAL, ½ oz. with separate dropper.	pkg	1
	91028	BAL ointment, ¾ oz. tube	tubes	2
	91050	Calamine lotion, NF approximately 2 fluid oz.	pkg	1
	91058	Chloroform, USP approximately 2 fluid oz.	pkg	1
	91091	Eye and nose drops, ½ oz., with separate dropper.	pkg	1
	91172	Phosphorus burn set	set	1
	91187	Protective ointment, CWS; 3 oz	tube	1
	92118	Pad, cotton, approximately 1¼ x 2 in	pkg	1
2.	97767	Kit, treatment, gas casualty:		
	97768	Kit, treatment, gas casualty, container	ea	1
	1K24810	Eye, solution, BAL, ½ oz	pkg	2
	1K76525	Sodium, sulamyd, 1 gram 25	pkg	1
	10690	Amyl nitrite, USP 5 minim. amp. 10	pkg	4
	74930	Soap, white floating	bar	2
	91027	Amyl salicylate, 8 oz	tin	3
	91028	BAL ointment, two ¾-oz. tubes	set	6
	91048	Calamine concentrate, two ¾-oz. tubes	pkg	2

Medical Department item No.	Item	Unit	Quantity
2. 91058	Chloroform, USP approx. 2 fluid oz	bottle	2
91073	Copper sulfate powder, approximately 35 gm.	pkg	1
91091	Eye and nose drops, ½ oz	bottle	4
91163	Ophthalmic discs, fluorescein and atropine sulfate.	box	1
91165	Petrolatum, USP, two ⅝-oz. tubes	pkg	7
91075	Copper sulfate solution, 5%, approximately 1.75 oz. with small forceps.	pkg	1
91187	Protective ointment, CWS	tube	4
91211	Sulfanilamide, crystalline, USP, 5 grams in sterile individual double-wrapped envelope—5 envelopes.	pkg	4
91213	Sulfathiazole ointment, 5%, two ⅝-oz. tubes.	pkg	7
99117	Bottle, plastic, 4 fluid oz	ea	1
99310	Kit, water testing, screening	ea	1
3. 97756	Set, gas casualty, M–2:		
97787	Pack board	ea	1
97922	Unit medical equipment pack, case empty.	ea	1
97767	Kit, treatment, gas casualty	ea	1
97923	Unit medical equipment pack, insert, empty.	ea	2
99030	Apron, impermeable	ea	6
99263	Gloves, impermeable	ea	6
4. 97758	Set, gas casualty, aprons and gloves:		
97470	Blanket set, small, case, empty	ea	1
99030	Apron, impermeable	ea	20
99263	Gloves, impermeable	ea	20
5. 93640	Oxygen therapy apparatus, closed circuit, Boothby-Lovelace.		
6. 1K24808	Eye ointment BAL: 3-gm. tube individual issue.		
7. 98156	Gas casualty set, veterinary, chest, "A", complete:		
98158	Gas casualty set, veterinary, chest empty, equipped with insert, plywood, No. 2 (97761) and cover, canvas, dental pack chests (97737).		
13722	Potassium permanganate, USP	5 lb	4
74590	Brush, scrub	ea	5
91053	Calcium hypochlorite	3¾ lb	4
99030	Apron, impermeable	ea	1
99263	Gloves, impermeable	pr	6
8. 98155	Gas casualty set, veterinary, case, aprons:		
97515	Case, tent pin	ea	1
99030	Apron, impermeable	ea	8
99140	Bucket, canvas	ea	4

Medical Department item No.	Item	Unit	Quantity
9. 98157	Gas casualty set, veterinary, chest, "B," complete:		
98158	Gas casualty set, veterinary, chest, empty, equipped with insert, plywood, No. 2 (97761) and cover, canvas, dental pack chests (97737).	ea	1
10110	Acid, boric, USP	lb	1
11085	Butyn, NNR, 3 gr. hypo. tab	10	2
11615	Cupric sulfate, USP	lb	1
12135	Gentian violet powder, medicinal	lb	1
14150	Sodium bicarbonate, USP	lb	2
14635	Sulfanilamide, USP, powder	lb	4
1K24810	Eye solution, BAL, ½ oz	pkg	6
55970	Syringe, water	ea	5
74590	Brush, scrub	ea	5
74930	Soap, white, floating	bar	10
99030	Apron, impermeable	ea	1
99140	Bucket, canvas	ea	6
99263	Gloves, impermeable	pr	4

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*Number denotes Medical Department Supply Catalog Number.